1979

Gene flow versus selection in the maintenance of certain polymorphisms in Mus musculus.

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GENE FLOW VERSUS SELECTION IN THE MAINTENANCE OF CERTAIN POLYMORPHISMS IN *MUS MUSCULUS*

by

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A Thesis
Submitted to the Faculty of Graduate Studies through
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ABSTRACT

An examination of house mouse (Mus musculus) populations collected from corn cribs in southwestern Ontario revealed evidence for the widespread distribution of polymorphism at 7 of 12 loci controlling renal enzymes (Id-1, Mod-1, Got-2, Dip-1, Gpd-1, Pgm-1, and Pgm-2); 5 loci were monomorphic (Got-1, Ipo-1, Trip-1, Mpi-1, and Ldh-1). Also, two loci, Pgm-1 and Pgm-2, controlling erythrocytic enzymes, were found variable. At each of these two loci, a previously unreported allele was detected. These alleles, $Pgm\text{-}1^{e}$ and $Pgm\text{-}2^{c}$, were found at very low frequency, 0.0247 and 0.0049, respectively.

The populations were measured for genic heterozygosity based on 12 loci, with the average heterozygosity established as 0.0886, a value well within the range of other investigations of Mus and other species.

Upon calculation of genetic distance, there appeared to be a slightly significant correlation between geographic and genetic distances, suggesting that random genetic drift within the populations was being counteracted by other forces, perhaps selection and/or migration.

Variance analysis was performed. Wahlund's variance ranged from zero to 0.4800 with the mean at 0.0624. Furthermore, genotypic ratios showed a significant departure from a Hardy-Weinberg equilibrium at 5 of 7 polymorphic loci.

Both the departures from Hardy-Weinberg equilibrium and the ecological data are consistent with population subdivision and should not be observed in a panmictic situation.

Migration rate and effective population size estimates were calculated. A mean migration rate of 0.2981, based on an effective population size of 10, could maintain the allelic frequencies found and alone could effectively oppose random genetic drift. Therefore, there was no need to invoke natural selection to account for the results.

In summary, the various results combined with the information derived from observation of the populations suggested the populations under consideration were subdivided and the uniformity in allelic frequencies could be explained by gene flow.

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I INTRODUCTION

The existence of genetic polymorphisms has been well documented for most species (Allison, 1955; Cain and Sheppard, 1950; Ford, 1953; Harris, 1969; Lamotte, 1959; Selander et al., 1970) and populations of Mus musculus prove to be no exception (Nichols and Ruddle, 1973; Petras, 1967a, b; Ruddle et al., 1969; Selander et al., 1969). This leads to one of the key questions in evolutionary biology and the main focus of this study: What are the relative roles of mutation, migration, and selection in maintaining genetic polymorphisms?

Genetic polymorphism, defined as the occurrence in the same population of two or more alleles at one locus, each with appreciable frequency (Cavalli-Sforza and Bodmer, 1971), has been studied extensively in wild populations of Mus (Petras et al., 1969; Nichols and Ruddle, 1973; Selander et al., 1969).

Considerable attention has also been paid to allelic frequency variance for many species, including man (Nei and Imaizumi, 1966a, b) and mouse (Selander, 1970a, b).

While allelic frequency and variance data are valuable measures in themselves, they may be further used as the basis for estimates of migration rate and effective population size, both of which are difficult to measure in natural populations

of Mus musculus. Generally, migration between Mus populations has been reported as infrequent (Southern and Laurie, 1946; Brown, 1953; Rowe et al., 1963) and breeding unit size as small (Lewontin and Dunn, 1960; Petras, 1967a).

The present study has specific advantages for dealing with the aforementioned problem. Firstly, animals of the species Mus musculus are excellent research organisms. The anatomy, physiology, ecology, and genetics of the house mouse are quite well established (Gruneburg, 1952; Green, 1966). Sexual maturity occurs at approximately the 6 or 7 week stage of life and the gestation period is about 21 days, making the house mouse an excellent candidate for breeding studies. Furthermore, inbred lines of mice have been developed and their chromosomes extensively mapped.

Secondly, Mus populations of southwestern Ontario lend themselves well to the solving of the problem under consideration in that they are discontinuous. Corn cribs, from which the mice were collected, are natural isolates, pockets of optimal habitat. Because of this, any one or a combination of genetic drift, mutation, migration, and selection may affect the allelic frequencies in these isolated populations, resulting in genetic differentiation.

Twelve loci were studied in the population of Mus under consideration; 7 were polymorphic. The data from these polymorphic loci will be analyzed in attempt to answer the main question of this investigation. As measures of variability, genic heterozygosity and genetic distance will be examined.

Furthermore, migration rate and breeding unit size will be determined to gain insight into the population structure of the populations considered. Finally, with information on allelic frequency, variance, migration rate, and effective population size, an attempt will be made to determine the roles that mutation, migration, and selection must have to explain the observed genetic variability.

II GENERAL METHODS

A Sampling Procedures

Mus musculus, or the common house mouse was collected from two counties of southwestern Ontario: Essex and Kent. This area is just east of the Detroit River, between Lakes St. Clair and Erie and encompasses two major urban centres, Windsor and Chatham, separated by about 50 miles. The 8 sampling regions are listed in Table 1 and indicated in Figure 1. Mice were collected from 52 corn cribs and two barns located on the farms throughout the study area.

The mice were collected when the corn cribs or barns were being emptied. At any one farm, this occurred over no more than three days. Normally, the samples were collected from various farms of the region throughout the spring and summer months.

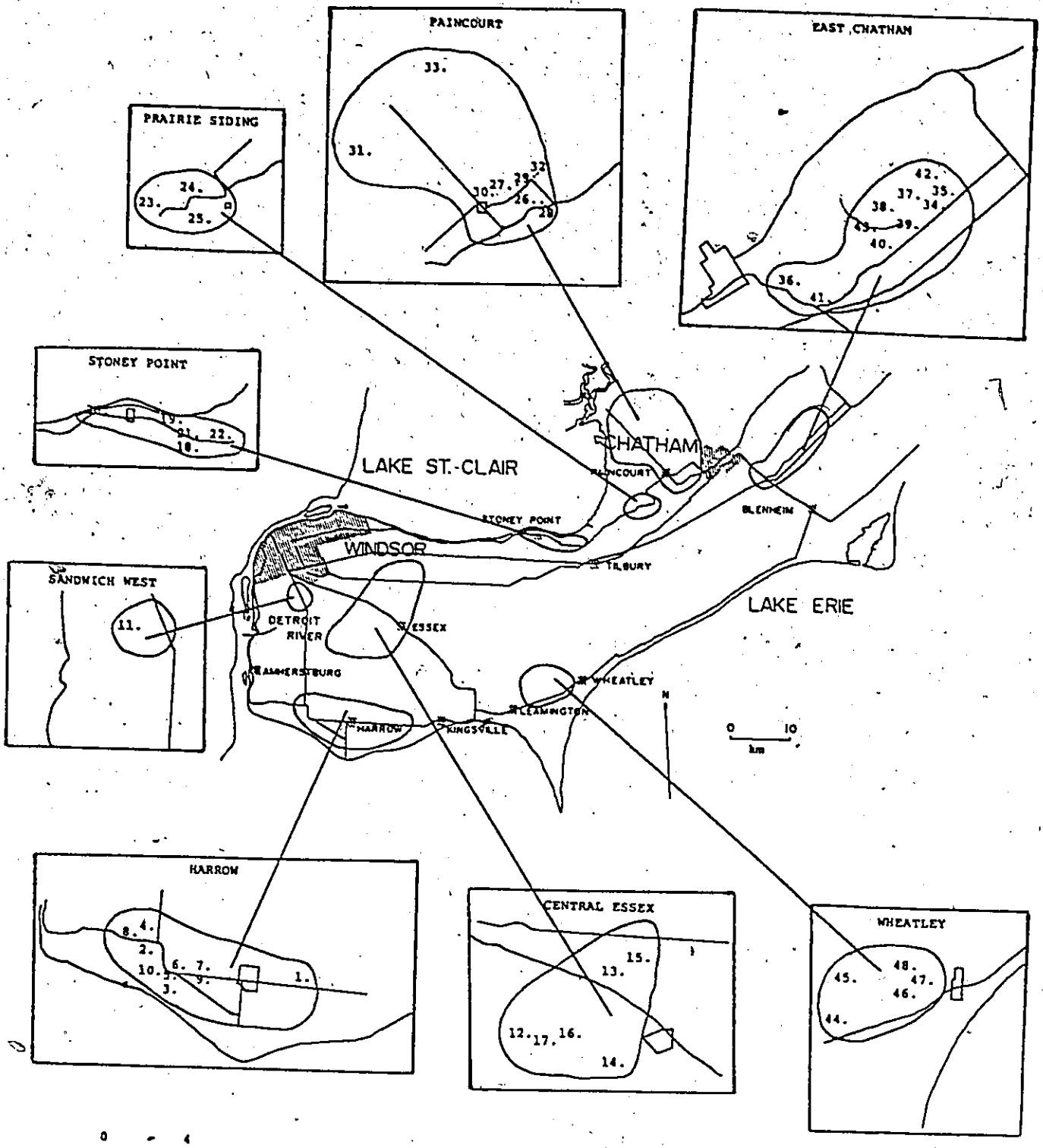
The trapping procedure basically consisted of setting up a hardware cloth barrier about a yard from the concrete base of the corn crib and placing Sherman live-traps within the enclosed area. This system was quite efficient, usually resulting in the capture of over 75 percent of the total population, thereby reducing the sampling variance. Hawkeswood (1975) and Topping (1975) have described crib sites and sampling procedures in more detail.

The animals used in this investigation were collected

Table 1: Regions and farm sites sampled in southwestern Ontario.

REGIONS	FARM SITES
Harrow	1. Arner
	2. Bennett
	3. Bondy
	4. Hutchins
	5. Langois
	6. D. Martin
	7. K. P. Martin
	8. Parkes (E. and W.)
	9. Pidgeon
	10. Richardson
Sandwich West	11. Walters
Central Essex	12. Chapo
	13. Maitre (N. and S.)
	14. McKim
	15. Price (N. and S.)
	16. Rocheleau East
	17. Rocheleau West
Stoney Point	18. Baillargeon
	19. Comartin (E. and L.)
	20. Damphouse
	21. Houle
	22. Nussey
Prairie Siding	23. Caron
	24. Coulet
	25. Pinnsonneault
Paincourt	26. Belanger
	27. G. Belanger
	28. Chenick
	29. Faubert
	30. Gagnier
	31. Ouellette
	32. Roy III
	33. Roy XII
East Chatham	34. Bodner
	35. Brown
	36. Communication Road
	37. Johnston
	38. Lenover
	39. Nash
	40. Nash Gravel Pit
	41. Snobdelen
	42. Thatcher
	43. Van K
Wheatley	44. James
	45. Leslie
	46. C. Wilkinson
	47. G. Wilkinson
	48. N. R. Wilkinson

Figure 1: Map of trapping sites. The numbers correspond to the trapping sites listed in Table 1.



from 1973 to 1976.

Upon capture, all mice were brought to the laboratory, numbered, and a record of the sex, age, and farm source was kept. Over the following months, kidney and blood samples were obtained from the animals and tested for various biochemical markers.

B. Enzyme Systems and Electrophoretic Methods

Twelve biochemical markers were examined in the kidney homogenates of the mouse and two in hemolysates.

1. Isocitrate Dehydrogenase (IDH)

Three forms of IDH have been described by Henderson (1965). Two of these are mitochondrial enzymes; the third is an NADP-dependent supernatant enzyme (IDH-1). Only the latter was used in the present study. Henderson (1965) described patterns controlled by three alleles, *Id-1*^{a}, *Id-1*^{b}, and *Id-1*^{c} at the *Id-1* locus. The first two alleles are found both in inbred strains and natural populations, while the third is found only in natural populations. *Id-1* is on chromosome 1 (Chapman *et al.*, 1970; Hutton and Roderick, 1970).

In the present study electrophoretic separation was carried out on starch gel in a 0.03M citrate-phosphate buffer of pH 7.0 (Henderson, 1965). The gel was then incubated in a staining mixture of 0.5M isocitric acid, phenazine methosulfate (PMS), and nitro-B-tetrazolium (NBT) (Henderson, 1965).

The patterns observed are shown in Figure 2. The inbred strain C57BL/6J is homozygous for Id-1^a and DBA/2J for Id-1^b. The heterozygote has three bands.

2. Nicotinamide Adenine Dinucleotide Phosphate (NADP) - Malic Enzyme (MOD)

There are two genetically different forms of MOD, the supernatant and mitochondrial (Henderson, 1966). It is the supernatant form, MOD-1, controlled by locus Mod-1 on chromosome 9 (Henderson, 1966; Hutton and Roderick, 1970), that was found to be polymorphic in natural populations (Shows et al., 1970).

The buffer system and staining mixture used were those of Shows and Ruddle (1968): 0.008M tris (hydroxymethyl)aminomethane (tris) and 0.003M citrate buffer of pH 6.3 and 0.5M malic acid, PMS, and NBT, respectively. The results of electrophoresis are shown in Figure 3. The inbred strain DBA/2J is homozygous for Mod-1^a and C57BL/6J for Mod-1^b. The heterozygote has five bands.

3. Glutamate Oxaloacetate Transaminase (GOT)

Two forms of GOT have been separated by electrophoresis, a supernatant (GOT-1) and a mitochondrial (GOT-2) form (Wilson and Siperstein, 1959a, b; Fleisher et al., 1960; Nisselbaum and Bodansky, 1964; DeJimenez et al., 1967).

The supernatant form, GOT-1 is monomorphic in inbred strains of mice (Nichols and Ruddle, 1973), but a variant

Figure 2: Electrophoretic patterns for the NADP-supernatant form of isocitrate dehydrogenase (IDH-1).

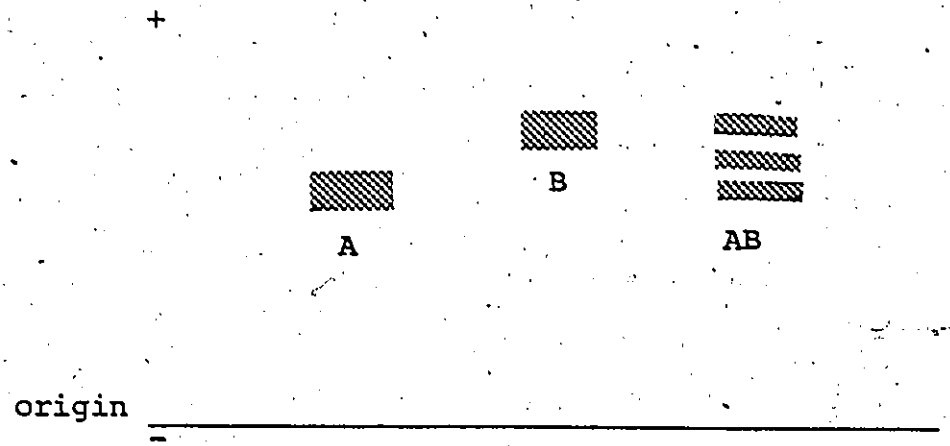
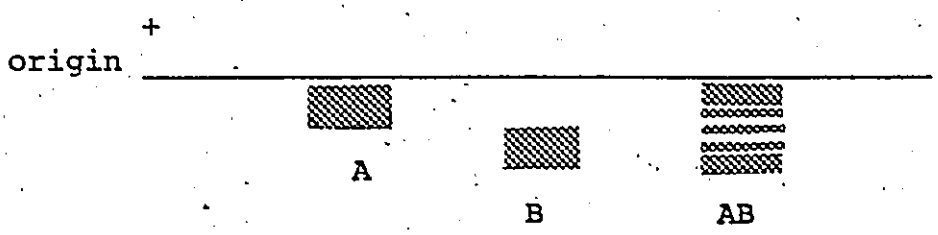


Figure 3: Electrophoretic patterns for the NADP-supernatant form of malic enzyme (MOD-1).



form, Got-1^b, was found in the Asian house mouse, Mus castaneus (Chapman and Ruddle, 1972). The linkage of Got-1 has not yet been determined. Got-1 was found monomorphic (i.e. all mice were homozygous for Got-1^a) in the natural populations examined.

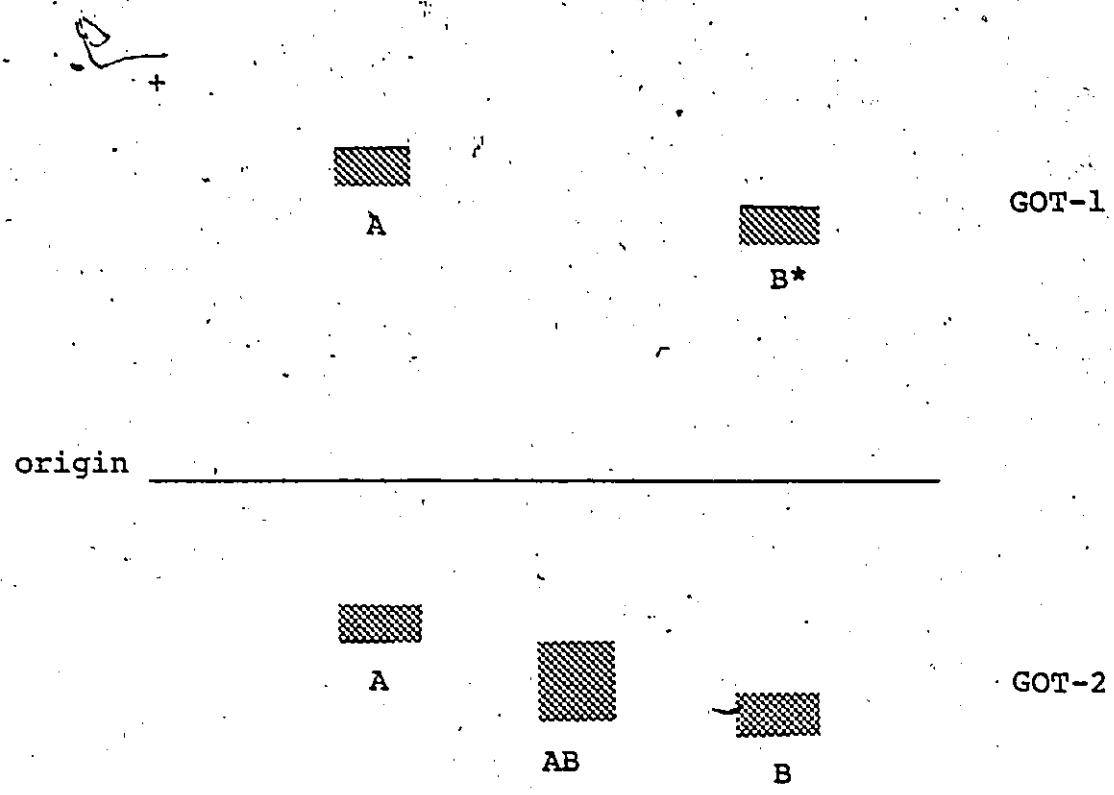
Two electrophoretic variants have been found for GOT-2 in the populations studied. These are similar to those described by DeLorenzo and Ruddle (1970). They are controlled by alleles Got-2^a and Got-2^b. The locus Got-2, has been found on chromosome 8 (DeLorenzo and Ruddle, 1970).

A buffer system modified from that described by Nichols and Ruddle (1973) was used. It was composed of 0.22M tris and 0.086M citric monohydrate of pH 8.6 and offered better resolution and separation of bands than the systems outlined by DeLorenzo and Ruddle. The staining mixture employed was the same as that used by DeLorenzo and Ruddle (1970) and consisted of L-aspartic acid, α-ketoglutaric acid, and fast blue BB. The electrophoretic patterns for GOT are shown in Figure 4. C57BL/6J is homozygous for Got-1^a and Got-2^b; the inbred strain SWR/J is homozygous for Got-2^a. Mus castaneus is homozygous for Got-1^b.

4. Peptidases

Five distinct peptidases in mouse erythrocytes have been discovered (Lewis and Truslove, 1969). Using a 0.9M tris, 0.2M ethylenediamine-tetraacetate acid disodium salt (EDTA), and 0.5M borate buffer of pH 8.6 (Nichols and Ruddle, 1973),

Figure 4: Electrophoretic patterns for the supernatant and and mitochondrial forms of glutamate oxaloacetate transaminase (GOT-1 and GOT-2, respectively). * GOT-1B band has so far been found only in Mus castaneus.



four different peptidases in kidney tissue were distinguishable: dipeptidase-1 (DIP-1), dipeptidase-2 (DIP-2), tripeptidase-1 (TRIP-1), and peptidase-S (PEP-S).

The staining mixture used was slightly modified from that of Nichols and Ruddle (1973). Crotalus adamanteus venom and 0.1M manganese chloride were added to each mixture (Lewis and Truslove, 1969). L-leucyl-L-tyrosine was the substrate used for DIP-1, DIP-2, and PEP-S, and L-leucylglycylglycine was used for TRIP-1. The dye was o-diansidine.

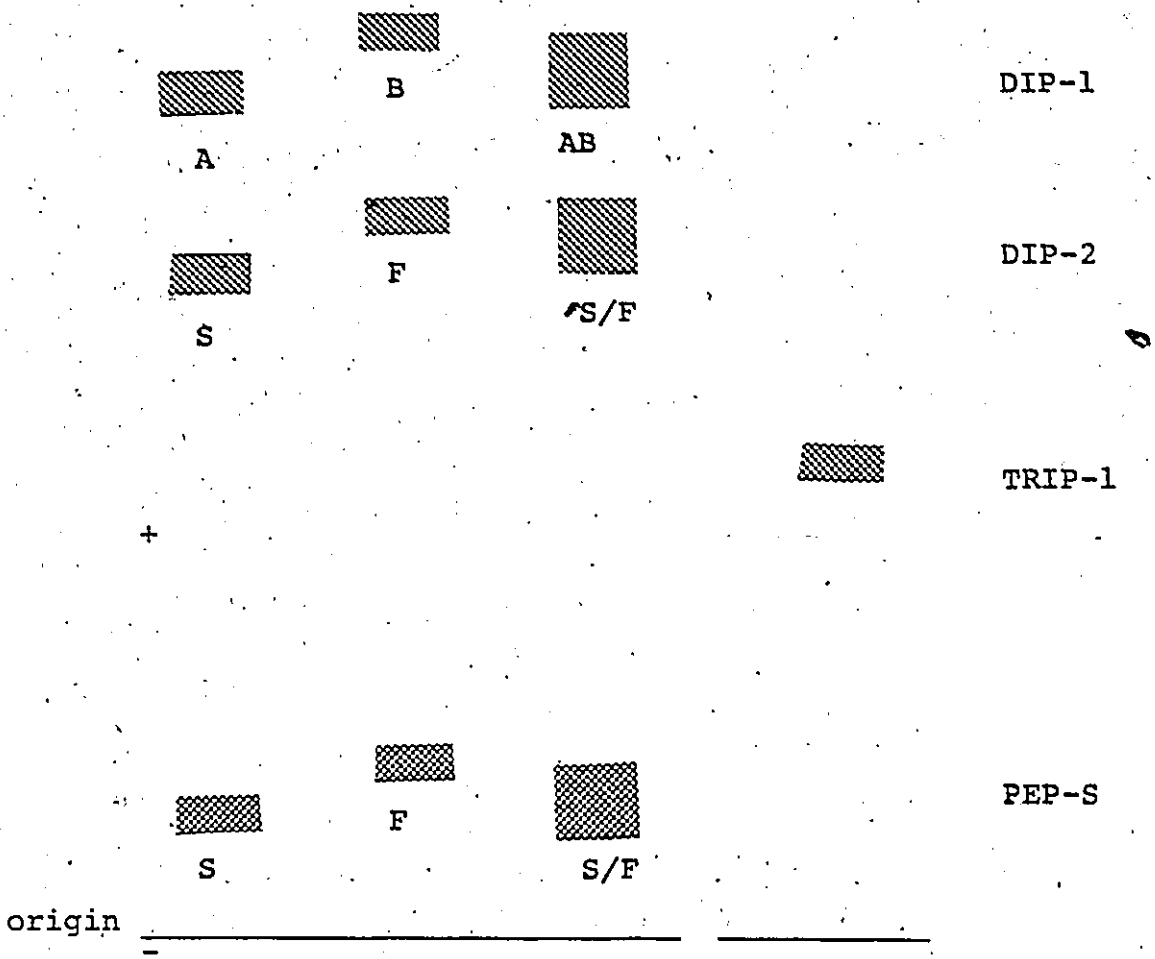
Three zones were resolved on one starch gel, with DIP-1 furthest from the origin, PEP-S closest, and DIP-2 intermediate to both. TRIP-1 was resolved on another gel. These electrophoretic results are shown in Figure 5.

DIP-1 is the only peptidase that has been described as polymorphic in mice (Nichols and Ruddle, 1973). The two variant forms are Dip-1^a and Dip-1^b. Dip-1 is on chromosome 1 with Id-1 (Chapman et al., 1970). C57BL/6J is homozygous for Dip-1^a and the inbred strain SJL/J for Dip-1^b.

DIP-2 has been found nonvariant in inbred mice (Nichols and Ruddle, 1973). In the natural populations under consideration, fast and slow bands were found, however, resolution and separation of the bands was not satisfactory, resulting in inconsistent typing. The linkage of Dip-2, as yet, has not been determined.

TRIP-1, as reported by Nichols and Ruddle (1973), was also nonvariant in the wild mice examined. The linkage of this locus has not been determined.

Figure 5: Electrophoretic patterns for four peptidases: dipeptidase-1 (DIP-1); dipeptidase-2 (DIP-2); tripeptidase-1 (TRIP-1); and, peptidase-S (PEP-S).



TRIP-1, as reported by Nichols and Ruddle (1973), was also nonvariant in the wild mice examined. The linkage of this locus has not been determined.

PEP-S is also not polymorphic in inbred mice and its linkage is still unknown (Nichols and Ruddle, 1973). Electrophoretic patterns resulted in inconsistencies in typing.

5. Glucose-6-phosphate Dehydrogenase (G6PD)

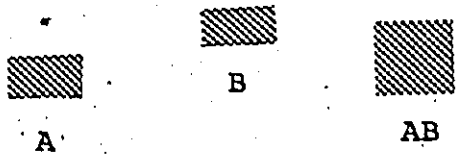
Two variant forms of G6PD have been reported in inbred mice, Gpd-1^a and Gpd-1^b (Ruddle et al., 1968, and both were found in the populations studied. Gpd-1 has been mapped on chromosome 4 (Ruddle et al., 1968).

For electrophoresis, a 0.9M tris, 0.02M EDTA, and 0.5M borate buffer of pH 8.6 (Ruddle et al., 1968) was used. Staining was done in a mixture consisting of 0.5M glucose-6-phosphate, NBT, and PMS (Nichols and Ruddle, 1973). The electrophoretic patterns for G6PD are shown in Figure 6. C57BL/6J is homozygous for Gpd-1^a and DBA/2J for Gpd-1^b.

6. Phosphoglucomutase (PGM)

The two major zones of electrophoretic activity designated for PGM are PGM-1 and PGM-2. For each of these regions, two electrophoretic phenotypes have been described in inbred and wild mice: Pgm-1^a and Pgm-1^b; Pgm-2^a and Pgm-2^b Shows et al., 1969; Chapman et al., 1970; Miner and Wolfe, 1972; Nichols and Ruddle, 1973). In the present study two alleles were detected: Pgm-1^a and a previously undescribed allele,

Figure 6: Electrophoretic patterns for one form of glucose-6-phosphate dehydrogenase (GPD-1).



Pgm-1^{e}. For the *Pgm-2* locus, three alleles were detected: *Pgm-2*^{a}, *Pgm-2*^{b}, and a previously undescribed allele, *Pgm-2*^{c}. The two new alleles will be described in greater detail later.

Pgm-1 has been assigned to chromosome 5 (Hutton and Roderick, 1970) and *Pgm-2* to chromosome 4 (Chapman *et al.*, 1970).

The buffer system used was the one described by Spencer *et al.* (1964): 0.1M tris, 0.1M maleic acid, 0.01M EDTA, and 0.01M magnesium chloride of pH 7.6. The stain was somewhat modified from Spencer *et al.* (1964):

0.2M tris-HCl, pH 8.0 (50 ml)
glucose-1-phosphate (0.4 g) (substrate)
0.25M magnesium acetate (6ml)
NADP (0.02 g)
crystalline glucose-6-phosphate dehydrogenase (100λ)
glucose-1,6-diphosphate (0.005 g)
PMS (0.005 g)
NBT (0.02 g).

The electrophoretic separation of the two PGM zones is shown in Figure 7. C57BL/6J is homozygous for *Pgm-1*^{a} and *Pgm-2*^{a}; DBA/2J for *Pgm-1*^{b}; and, the inbred strain SM/J for *Pgm-2*^{b}.

Two other zones of activity appeared on most starch gels stained for PGM. These zones, previously undescribed, designated as "PGM-3" and "PGM-4", were found intermediate to the PGM-1 and PGM-2 zones on the starch gels, with "PGM-3" zone migrating more anodally than the "PGM-4" (Figure 8). Three patterns in wild animals were found for the "Pgm-3"

Figure 7: Electrophoretic patterns for two forms of phosphoglucomutase (PGM-1 and PGM-2).

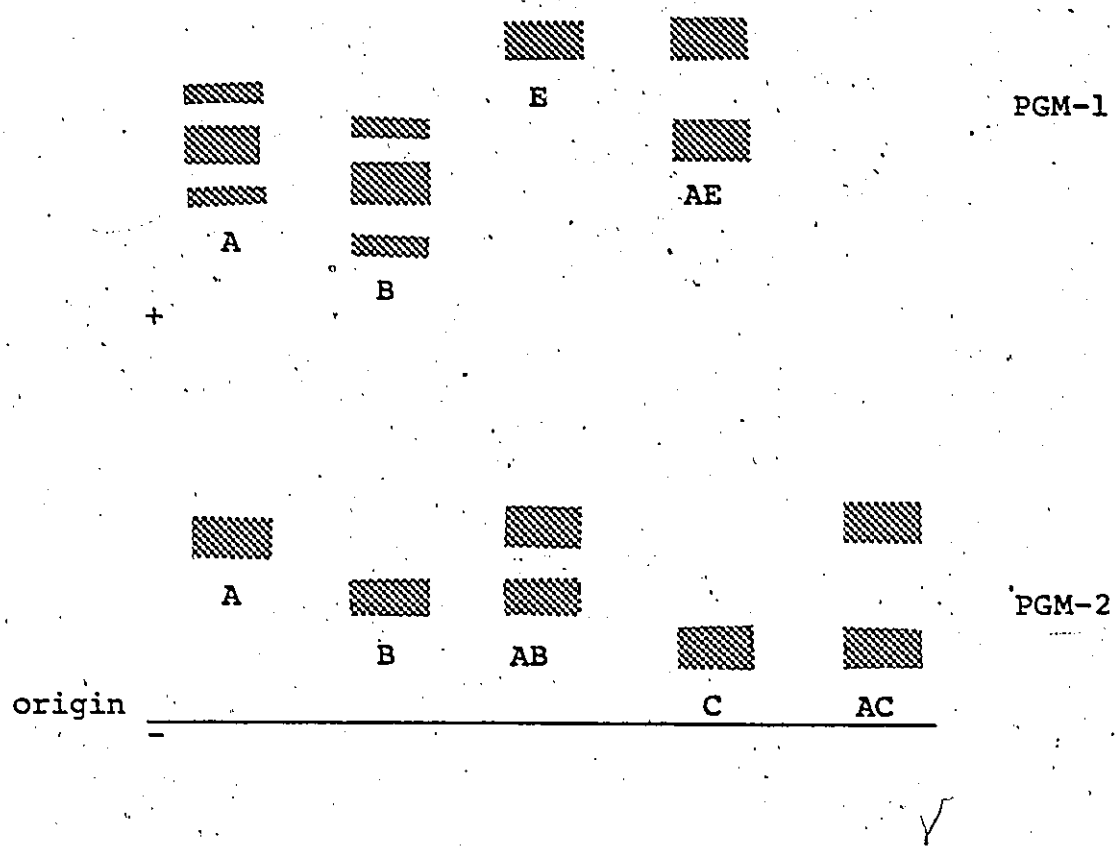
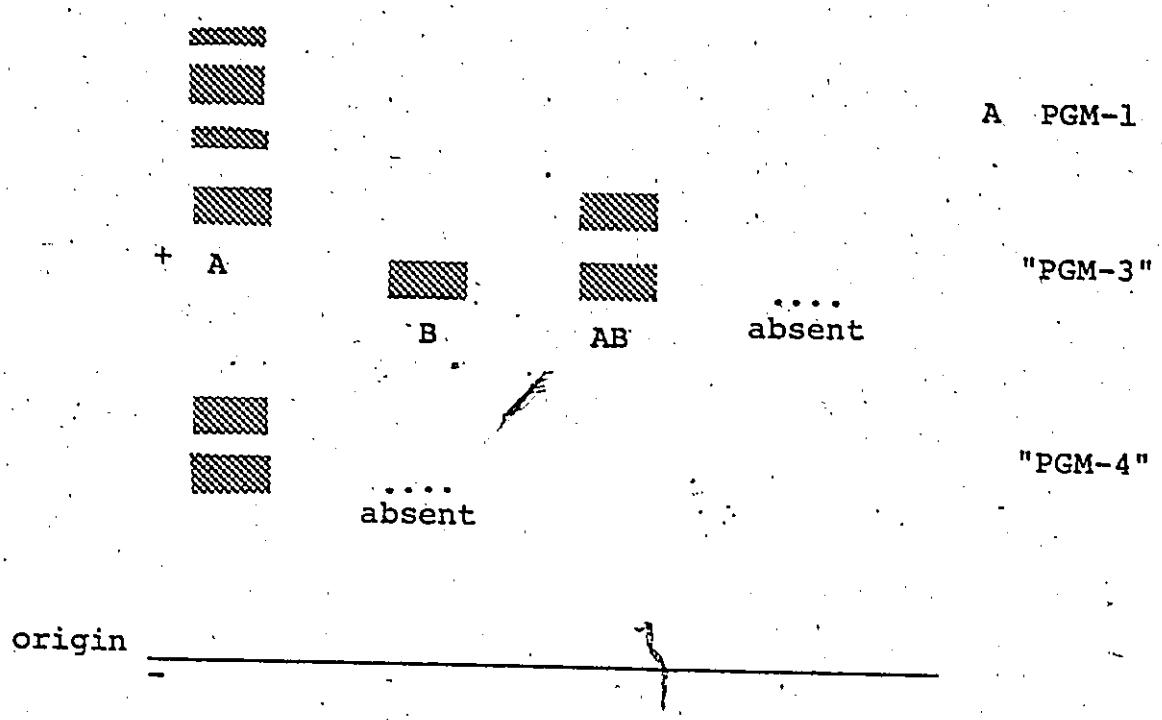


Figure 8: Electrophoretic patterns for two new zones of phosphoglucomutase activity ("PGM-3" and "PGM-4").



zone: a single "PGM-3A" band, slightly slower than PGM-1A; a "PGM-3B" band, slower migrating than "PGM-3A"; and, a double band, "PGM-3AB". Also, in several cases there was no indication of the "PGM-3" zone.

The pattern for the "PGM-4" zone was either the presence of a single band, slower than "PGM-3B" or its complete absence.

These two new zones of electrophoretic activity were consistently detected in the wild populations and showed variation in their phenotype. Unfortunately, reading these patterns was difficult because the separation and resolution of these zones varied considerably for unknown reasons.

7. Indolphenol Oxidase (IPO)

Two forms of IPO, IPO-A and IPO-B, have been detected on electrophoresis with no inbred variants reported for either form (Nichols and Ruddle, 1973). Selander and Yang (1969) have reported two electrophoretic forms for the anodally migrating IPO region in Danish house mice. No variants were found in the present study.

A buffer system of 0.9M tris, 0.02M EDTA, and 0.5M borate of pH 8.6 was used. The staining mixture consisted of MTT tetrazolium and PMS. The buffer and stain were those of Nichols and Ruddle (1973).

8. Lactate Dehydrogenase (LDH)

Five zones of activity have been described for LDH

(Martin and Petras, 1971), with LDH-A the slow migrating subunit and LDH-B the fast migrating subunit. A 0.9M tris, 0.02M EDTA, and 0.5M borate buffer of pH 8.6 was used. The stain consisted of sodium-L-lactate, PMS, and NBT. Both buffer and stain were those described by Shows et al. (1968). Polymorphism has not been reported for either LDH-A or LDH-B in inbred or wild mice (Nichols and Ruddle, 1973) and was also not detected at any of the LDH zones in the kidney samples studied.

Shows and Ruddle (1968) found the regulation of LDH-B expression in mouse erythrocytes to be controlled by a single autosomal locus, Ldr-1, mapped to chromosome 6 (Hutton and Roderick, 1970).

Several loci may control LDH expression, however, for the purposes of this present study, only one locus, Ldh-1, will be considered.

9. Mannosephosphate Isomerase (MPI)

Two electrophoretic forms of MPI, discovered in inbred strains, have been reported: MPI-1A and MPI-1B (Nichols et al., 1973). The populations examined in the present study, however, were monomorphic (i.e. all mice were homozygous for $Mpi\text{-}1^{b}$).

The single autosomal locus, Mpi-1, controlling the two alleles, has been mapped on chromosome 9 (Nichols et al., 1973).

Electrophoresis on starch gel in a 0.9M tris, 0.02M

EDTA, and 0.5M borate buffer of pH 8.6 was followed by a staining mixture of D-mannose-6-phosphate, MTT tetrazolium, and PMS, as described by Nichols et al. (1973). The electrophoretic separation of MPI is shown in Figure 9. The inbred strain MA/J is homozygous for Mpi-1^a and C57BL/6J for Mpi-1^b.

C General Methods for Electrophoresis and Tissue Preparation

The electrophoretic apparatus and method of preparing the 14 percent starch gels (Connaught Laboratories, Toronto) were basically those described by Biddle and Petras (1967). Horizontal electrophoresis was carried out in a cold room at temperatures of four degrees centigrade (^oC) for 18 hours, at approximately four volts per centimetre across the gel. The gels were then sliced in two horizontally and stained for about two hours at 37^oC in all cases. Chemicals used in buffers and sodium-L-lactate were supplied by Fisher Scientific Company; all other chemicals were supplied by Sigma Chemical Company.

The blood collecting procedure from the suborbital sinus and hemolysate preparation were similar to those described by Biddle and Petras (1967).

For kidney homogenates, the kidneys were removed by incision through the dorsum after the animal was killed by cervical dislocation. The kidneys were homogenized in twice their weight of distilled water by a Polytron tissue homogenizer at setting four for 30 seconds in ice. Next, the sample

Figure 9: Electrophoretic patterns for one form of mannosephosphate isomerase (MPI-1).

was frozen at -6°C and thawed three times and then centrifuged at 27,000 X gravity (g) in a RC2-B (Sorvall) for one hour. The sample was then stored at -6°C until time of use, when it was again centrifuged at 27,000 X g for one hour before electrophoresis.

III KIDNEY ENZYME STUDY

A Loci Studied Controlling Renal Enzymes

Genetic variation involving 12 renal enzymes of the house mouse was studied electroporetically in samples of mice collected during the summers of 1973 and 1974. Based on a sample of over 200 mice, 5 of the 12 loci were found to be monomorphic in the mice captured in 1973. These included Got-1, Ipo-1, Trip-1, Mpi-1, and Ldh-1. With one exception, these monomorphic loci were not examined in the 1974 mice. That exception was Got-1, since the patterns controlled by this locus could be monitored simultaneously with those of a second form of the GOT enzyme, controlled by Got-2. The additional 467 mice captured in 1974 showed the Got-1 locus to remain monomorphic in that year.

The loci controlling the remaining enzymes included: Id-1, Mod-1, Got-2, and Dip-1, which had been studied in 1973 and again in 1974, and Pgm-1, Pgm-2, and Gpd-1, which were studied only in 1974 samples. These were all found polymorphic.

An attempt had been made to examine the 1973 mice for G6PD, however, because whole mice had been kept frozen for a period up to a year and this enzyme tends to deteriorate, the electrophoretic patterns of the kidney homogenates were not sufficiently clear to type with consistency. In the

1974 samples, the kidneys were removed immediately after the mice were killed. The electrophoretic patterns of these samples were much clearer and could be typed with confidence.

Similar, but not as extensive deterioration was seen in IDH and MOD from kidney homogenates of frozen mice, and therefore, the zymograms obtained were typable.

B Allelic Frequencies

A summary of the allelic frequencies at all polymorphic loci is given in Table 2.

All but one sample had two alleles present at the locus controlling the supernatant NADP-dependent form of IDH. The exception occurred in a small sample from the Arner farm. The Id-1^a allele was the more common allele in nearly all the populations sampled and appears as the more common allele in the pooled data: 0.673 and 0.880, in 1973 and 1974, respectively. The increase in frequency of Id-1^a from 1973 to 1974 appears to be quite widespread.

Of the two alleles at the locus responsible for the supernatant form of NADP-dependent malic enzyme, Mod-1^a was found to be the more frequent in samples collected in both 1973 and 1974. All but three of 15 populations in 1973 and 10 of 29 in 1974 had two alleles; the exceptions had only Mod-1^a. Of the fixed populations, only two, Maitre South and Brown, had a sample size greater than 10 and only in one region, Paincourt, were all populations studied monomorphic and furthermore, monomorphic in both years. As with the

Table 2: The allelic frequencies and standard errors of the more frequent alleles at polymorphic loci in populations of Mus musculus. Only samples consisting of five or more mice from a particular locality are generally included. (SS - average sample size).

SITE / REGION	1973 SS	Id-1[a]	Mod-1[a]	Gpi-2[a]	Pip-1[b]	1976 SS	Id-1[a]	Mod-1[a]	Gpi-2[a]	Pip-1[b]	Gpd-1[b]	Pep-1[a]	Pep-2[a]
Arner	5	1.0000 ±0	0.7000 ±0.1449	0.9000 ±0.0949	0.9000 ±0.0949								
Bondy (crib)	5	0.6000 ±0.1549	0.3000 ±0.1449	0.8000 ±0.1265	0.8000 ±0.1265	38	0.8876 ±0.0411	0.9877 ±0.0225	0.8300 ±0.0299	0.3750 ±0.0533	0.6000 ±0.0548	0.9875 ±0.0124	1.0000 ±0
Bondy (barn)						5	1.0000 ±0	1.0000 ±0	0.7000 ±0.1449	0.8000 ±0.1265	0.2000 ±0.1265	1.0000 ±0	1.0000 ±0
D. Martin (crib)	4	0.2500 ±0.1531	0.8000 ±0.1265	1.0000 ±0	0.3750 ±0.1712	21	0.7895 ±0.0661	0.9118 ±0.0486	0.8636 ±0.0517	0.5609 ±0.0761	0.5682 ±0.0747	0.9773 ±0.0323	1.0000 ±0
Richardson						6	0.8750 ±0.1169	0.8000 ±0.1265	0.7857 ±0.1097	0.8571 ±0.0935	0.6429 ±0.1281	1.0000 ±1.0000	1.0000 ±1.0000
Bennett						11	0.9091 ±0.0631	0.8333 ±0.1076	0.8750 ±0.0675	0.7083 ±0.0928	0.3833 ±0.1006	1.0000 ±0	0.9583 ±0.0408
HARROW	17	0.6250	0.6750	0.8750	0.7188	86	0.8701	0.9338	0.8407	0.6319	0.5714	0.9890	0.9835
Walters						5	0.9000 ±0.0949	1.0000 ±0	0.7000 ±0.1449	0.8000 ±0.1265	0.2000 ±0.1549	0.8000 ±0.1265	1.0000 ±0
SANDWICH WEST						5	0.9000	1.0000	0.7000	0.8000	0.6000	0.8000	1.0000
Rochelseu East						7	0.9000 ±0.0949	1.0000 ±0	0.3750 ±0.1210	0.8125 ±0.0976	0.8125 ±0.0976	1.0000 ±0	1.0000 ±0
Rochelseu West						6	0.7000 ±0.1449	0.7500 ±0.1531	0.7500 ±0.1250	0.9167 ±0.0798	0.4167 ±0.1423	1.0000 ±0	1.0000 ±0
Maitre North	8	0.3750 ±0.1210	0.4375 ±0.1240	0.5000 ±0.1336	0.3714 ±0.1323	49	0.9388 ±0.0242	0.8776 ±0.0343	0.5400 ±0.0498	0.8300 ±0.0376	0.7128 ±0.0467	0.9900 ±0.0099	1.0000 ±0
Maitre South	32	0.4783 ±0.0737	0.6957 ±0.0678	0.6957 ±0.0749	0.7581 ±0.0679	24	0.9167 ±0.0199	1.0000 ±0	0.5417 ±0.0719	0.8750 ±0.0477	0.6304 ±0.0713	0.9375 ±0.0899	1.0000 ±0
Price North	13	0.6538 ±0.0933	0.7308 ±0.0870	0.7917 ±0.0829	0.4167 ±0.1006	12	0.9583 ±0.0408	0.8333 ±0.0761	0.4167 ±0.1006	0.9167 ±0.0564	0.6667 ±0.0962	0.9167 ±0.0564	1.0000 ±0
Price South						7	0.6429 ±0.1281	0.7500 ±0.1531	0.6429 ±0.1281	1.0000 ±0	0.7143 ±0.1207	1.0000 ±0	1.0000 ±0
CENTRAL ESSEX	47	0.5204	0.6900	0.6333	0.6111	110	0.9083	0.8969	0.5439	0.8738	0.6591	0.9693	1.0000
Damphouse	6	0.5833 ±0.1423	0.8750 ±0.0827	0.2000 ±0.1265	0 ±0								
E. Comartin	14	0.3357 ±0.0942	0.7813 ±0.0731	0.4583 ±0.1017	0.5417 ±0.1017	7	1.0000 ±0	1.0000 ±0	0.6250 ±0.1210	0.7500 ±0.1083	0.5625 ±0.1240	1.0000 ±0	1.0000 ±0
L. Comartin						7	1.0000 ±0	1.0000 ±0	0.6250 ±0.1210	0.6875 ±0.1159	0.5714 ±0.1323	1.0000 ±0	1.0000 ±0
Roule	127	0.6758 ±0.0293	0.7293 ±0.0272	0.5927 ±0.0312	0.5282 ±0.0317	43	0.8976 ±0.0344	0.8500 ±0.0399	0.8636 ±0.0366	0.6477 ±0.0509	0.6591 ±0.0505	1.0000 ±0	0.9773 ±0.0159
Baillargeon	6	0.5833 ±0.1423	0.8750 ±0.0827	0.5000 ±0.1768	0.5000 ±0.1768	5	0.9000 ±0.0949	0.8000 ±0.1265	1.0000 ±0	1.0000 ±0	0.5000 ±0.1581	1.0000 ±0	1.0000 ±0
SYDNEY POINT	155	0.6592	0.7465	0.5612	0.5136	66	0.9068	0.8871	0.7971	0.7101	0.6250	1.0000	0.9855
Finnsonneault	9	0.3889 ±0.1149	0.6000 ±0.1095	0.5625 ±0.1240	0.6875 ±0.1159								
PRAIRIE SIDING	9	0.3889	0.6000	0.5625	0.6875								
Caqnier	5	0.6000 ±0.1549	1.0000 ±0	0.5000 ±0.1581	0.5000 ±0.1581								
Belanger						6	0.6000 ±0.1547	1.0000 ±0	0.2857 ±0.1207	0.7143 ±0.1207	0.5000 ±0.1336	1.0000 ±0	1.0000 ±0
Faubert						6	0.9000 ±0.0949	1.0000 ±0	0.5833 ±0.1423	0.4167 ±0.1423	0.5833 ±0.1423	1.0000 ±0	1.0000 ±0
Roy III	8	0.5625 ±0.1240	1.0000 ±0	0.8571 ±0.0935	0.2857 ±0.1207								
FAINCOURT	16	0.6563	1.0000	0.7333	0.5667	14	0.6667	1.0000	0.4333	0.6333	0.5333	1.0000	1.0000
Communication Road						9	0.7778 ±0.0980	0.9375 ±0.0605	0.5556 ±0.1171	0.4444 ±0.1171	0.6111 ±0.1147	1.0000 ±0	1.0000 ±0
Snowdelen						12	0.8333 ±0.0761	0.9545 ±0.0444	0.4167 ±0.1006	0.6667 ±0.0962	0.5000 ±0.1021	1.0000 ±0	1.0000 ±0
Nash Gravel Pit						5	0.9000 ±0.0949	0.9000 ±0.0949	0.3000 ±0.1449	1.0000 ±0	0.6250 ±0.1712	1.0000 ±0	1.0000 ±0
Nash						7	0.8571 ±0.0935	0.7500 ±0.1250	0.6429 ±0.1281	0.9286 ±0.0688	0.6250 ±0.1712	0.9286 ±0.0688	1.0000 ±0
Brown	9	0.6000 ±0.1095	0.8636 ±0.0732	0.5625 ±0.1240	0.7500 ±0.1083	13	0.6250 ±0.0968	1.0000 ±0	0.5000 ±0.0981	1.0000 ±0	0.6923 ±0.0905	1.0000 ±0	1.0000 ±0
Johnston						16	0.7667 ±0.0772	0.9337 ±0.0456	0.5000 ±0.0884	0.8438 ±0.0642	0.6333 ±0.0880	1.0000 ±0	1.0000 ±0
Lenover						23	0.9130 ±0.0416	0.8810 ±0.0500	0.5217 ±0.0737	0.8913 ±0.0459	0.5750 ±0.0497	1.0000 ±0	1.0000 ±0
EAST CHATHAM	14	0.6667	0.9063	0.6538	0.6923	85	0.8118	0.9103	0.4941	0.8353	0.6079	0.9941	1.0000
C. Wilkinson						44	0.7727 ±0.0447	0.9762 ±0.0166	0.7333 ±0.0466	0.4212 ±0.0403	0.7061 ±0.0536	1.0000 ±0	0.9773 ±0.0155
M.R. Wilkinson						8	0.7500 ±0.1250	0.8333 ±0.1076	0.7500 ±0.1250	0.9167 ±0.0798	0.8333 ±0.1522	1.0000 ±0	1.0000 ±0
James						25	0.5111 ±0.0527	0.8696 ±0.0497	0.7800 ±0.0586	0.4200 ±0.0698	0.6600 ±0.0670	1.0000 ±0	0.9400 ±0.0336
Leslie						9	0.8889 ±0.0741	0.8571 ±0.0935	0.9000 ±0.0671	0.6000 ±0.1095	0.6500 ±0.1067	1.0000 ±0	1.0000 ±0
WHEATLEY						64	0.8274	0.9231	0.7674	0.6860	0.6892	1.0000	1.0000
Pooled for all regions	358	0.6231 ±0.0212	0.7518 ±0.0182	0.6107 ±0.0221	0.5615 ±0.0225	451	0.8796 ±0.0118	0.9131 ±0.0100	0.6492 ±0.0134	0.7516 ±0.0141	0.6767 ±0.0163	0.9872 ±0.0037	0.9893 ±0.0034

Id-1^a allele, a slight increase (0.752 to 0.913) was seen in the frequency of the Mod-1^a allele from 1973 to 1974.

The locus controlling the mitochondrial form of GOT, GOT-2, also had two alleles in all samples but two. In the latter, the small sample size could account for the absence of one allele. Got-1^a was the more frequent allele in all but a few small samples and in the pooled data. There was no significant change in the pooled frequency from one year to the next.

Of the two alleles, Dip-1^a and Dip-1^b at the Dip-1 locus, the latter was the more common, however, both were present in nearly all populations sampled. If fixation occurred, which it did in three populations, it was always Dip-1^a, the less common allele, that was eliminated. The overall frequency of Dip-1^b allele rose slightly from 0.564 in 1973 to 0.752 in 1974.

The frequencies of the two alleles, Gpd-1^a and Gpd-1^b, at the Gpd-1 locus, were quite similar in the populations sampled. Gpd-1^b appeared in all cases to be the more common.

Two different loci coding for the PGM enzyme, Pgm-1 and Pgm-2, were studied in the 1974 samples. Pgm-1^a was the more frequent allele at the Pgm-1 locus and in 20 of 29 populations sampled, it was the only detectable allele. The other allele, Pgm-1^e, which appears unreported in the literature was a relatively rare allele, having a frequency of only 0.013. At the Pgm-2 locus, again two alleles were found, Pgm-2^a and a previously undescribed allele, Pgm-2^c. Pgm-2^a

was found in most of the populations with Pgm-2^C occurring in animals from only four of the populations sampled. Its overall frequency was very low (0.011).

C Hardy-Weinberg Equilibrium Analysis

Table 3 summarizes the comparisons of observed phenotypes with those expected if the population was in a Hardy-Weinberg equilibrium for those samples which exceed 5 animals. All animals were, however, included in the pooled data for any given region.

Statistically significant departures of genotypic frequencies from a Hardy-Weinberg equilibrium have been rare, partly because of small sample sizes (Cavalli-Sforza and Bodmer, 1971). The current study has this same limitation and as a result, only those samples where the population size exceeds 20 should be taken seriously.

There are a number of causes of departures from Hardy-Weinberg equilibrium. In the current study, all departures from Hardy-Weinberg equilibrium expectations were the result of a numerical deficiency of heterozygotes. The commonly stated causes include: presence of a silent allele; errors in classification of phenotypes; positive assortive mating; selection against the heterozygote; and, population structuring.

There appear to be no indications that any of the first three are the likely explanations. After the examination of a large number of samples, a silent allele, if present for any of the loci studied, should have been detected in the

Table 3: A summary of sites at which samples consisting of five or more mice were collected, the size of each sample per locus studied, and the total number of mice per locus studied from each region. The asterisks refer to populations which significantly differ from a Hardy-Weinberg equilibrium (* - probability (P) less than 0.05; ** - P less than 0.01).

SITE/REGION	1973				1974						
	Id-1	Mod-1	Got-2	Dip-1	Id-1	Mod-1	Got-2	Dip-1	Pgm-1	Pgm-2	Gpd-1
Arner	5	5	5	5							
Bondy (crib)	5	6	5	5	34	31	40	40	40	40	40**
Bondy (barn)					4	4	5	5	5	5	5
D. Martin	4	5	4	4	19	17	22	22	22	22	22**
Richardson					4	5	7	7	7	7	7
Bennett					11	6	12	12	12	12	12
HARROW	16	20	16	16*	77**	68	91	91	91	91	91**
Walters					5	5	5	5	5	5	5
SANDWICH WEST					5	5	5	5	5	5	5
Rocheleau East					6	5	8	8	8	8	8
Rocheleau West					5	4	6	6	6	6	6
Maitre North	8	8	7	7	49	45**	50	50**	50	50	47*
Maitre South	23	23**	21	21	24	23	24	24	24	24	23
Price North	13	13	12	12	12	12	12	12	12	12	12
Price South					7	4	7	7	7	7	7
CENTRAL ESSEX	49	50**	45**	45**	109	97**	114	114**	114	114	110**

Table 3: continued

SITE/REGION	1973				1974						
	Id-1	Mod-1	Got-2	Dip-1	Id-1	Mod-1	Got-2	Dip-1	Pgm-1	Pgm-2	Gpd-1
Damphouse	6	8	5	5							
E. Comartin	14	16	12	12	6	6	8	8	8	8	8
L. Comartin					6	7	8	8	8	8	8
Houle	128	133**	124**	124**	39	40	44**	44	44	44	44**
Baillargeon	6	8	4	4	5	5	5	5	5	5	5
STONEY POINT	157	169**	147**	147**	59	62**	69*	69	69	69	68**
Pinnsonnealt	9	10	8	8							
PRAIRIE SIDING	9	10	8	8							
Gagnier	5	5	5	5							
Belanger					5	3	7	7	7	7	7
Faubert					5	5	6	6	6	6	6
Roy III	8	9	7	7							
PAINCOURT	16**	17	15	15*	12	9	15*	15	15	15	15

Table 3: continued

SITE/REGION	1973 Id-1	1973 Mod-1	1973 Got-2	1973 Dip-1	1974 Id-1	1974 Mod-1	1974 Got-2	1974 Dip-1	1974 Pgm-1	1974 Pgm-2	1974 Gpd-1
Communication Road					9	8	9	9	9	9	9
Snobdelen					12	11	12	12	12	12	12
Nash Gravel Pit					5	5	5	5	5	5	5
Nash					7	6	7	7	7	7	5
Brown	10	11	8	8	12	11	13	13	13	13	13
Johnston					15	15	16	16	16	16	15
Lenover					23	21	23	23	23	23	23
EAST CHATHAM	15	16	13	13	85*	78**	87	87**	87	87	84
C. Wilkinson					44	42	45	45	45	45	45
N. R. Wilkinson					6	6	6	6	6	6	5
James					25	23	25	25	25	25	25**
Leslie					9	7	10	10	10	10	10
WHEATLEY					84	78**	86	86**	86	86	85**

homozygous condition. It was not. Hence, if a silent allele does exist, it does so in a very low frequency and would not account for the outstanding deficiency of heterozygotes. Neither would errors in classification of phenotypes explain this deficiency since all electrophoretic results were rechecked carefully. Again, no evidence was seen to invoke positive assortive mating. One could postulate a relationship between certain enzymes and/or their products and olfactory cues, but there is no evidence for this, and furthermore it would be highly unlikely to invoke products of all 7 loci simultaneously.

The two other causes of departure from Hardy-Weinberg expectations, selection against the heterozygote and population structuring or inbreeding, are more likely candidates. The postulate of selection against the heterozygote is difficult to prove, but equally difficult to disprove. However, polymorphisms at the variable loci appear widespread geographically and persist in the overall region from year to year. Therefore, if indeed selection against the heterozygote was in effect, the elimination of the rarer allele should be expected in at least some populations, but this did not happen frequently during the time of this study. Thus, the explanation of population structuring remains as the most likely.

In fact, each population sampled may very well be subdivided, that is, each corn crib population may consist of a number of independent subpopulations, with random mating occurring within each subpopulation. Hardy-Weinberg equilibrium

may be maintained within any given subpopulation, subject to minor disturbances due to its finite size. But, upon pooling of these subpopulations for each corn crib, there may be an overall significant deficiency of heterozygotes. The same effect may hold for the data from the corn crib samples pooled for the regional population. When the corn crib populations, with different allelic frequencies, but each at Hardy-Weinberg equilibrium, are pooled there may be an overall deficiency of heterozygotes. This effect, known as Wahlund's principle, points out the variations between populations due to random genetic drift, may very well be the explanation for the significant deficiency of heterozygotes in the southwestern Ontario populations.

Table 3 shows that of the 10 populations in which the sample was equal to or exceeded 20 animals, 7 showed a significant departure from a Hardy-Weinberg equilibrium at at least one locus or 13 of 42 "population - locus - year" samples. Most of the larger samples (sample size greater than 40) showed departure from a Hardy-Weinberg equilibrium at more than one locus. Furthermore, when the samples from a given region were pooled, all of those which exceeded 40 mice showed deviations at at least two loci, most at three.

In pooled data for a region several different patterns of contribution to a significant chi-square value were seen:

(a) <u>A single large population contributed most to the pooled chi-square value</u>. Four cribs were sampled in

the Stoney Point region in both 1973 and 1974. Only the animals from the Houle population showed a significant deviation from Hardy-Weinberg expectations for the Dip-1 locus in the 1973 samples and Got-2 locus in the 1974 samples. The samples from the remaining three sites did not give a significant chi-square value. In this case, the deviation from Hardy-Weinberg equilibrium in the pooled sample of the region was primarily the result of a single sample.

(b) Not only did a single large sample contribute to the significant chi-square value, but so did the remaining samples when pooled. Six different corn cribs made up the Central Essex-1974 populations. The sample from only one of these, Maitre North, showed a significant deviation from Hardy-Weinberg equilibrium for three loci, Mod-1, Dip-1, and Gpd-1. An analysis of the remaining 5 samples showed that a significant chi-square value persisted. Similar conclusions were reached for samples of three other corn cribs: Maitre South-1973 (Mod-1); Houle-1973 (Mod-1 and Got-2); Houle-1974 (Gpd-1); and, James-1974 (Gpd-1). In these samples then, there appeared to be a significant deficiency of heterozygotes at two levels, within a corn crib population and between corn crib populations.

(c) No single samples deviated significantly from

Hardy-Weinberg equilibrium, but pooled they did. The East Chatham-1974 population was made up of animals from 7 different corn cribs. A significant deviation from Hardy-Weinberg equilibrium was seen only in the pooled data of the region for three loci, Id-1, Mod-1, and Dip-1, and not in any of the contributing samples. Similar results for some of the loci were found for most regions, four of which, with a sample size greater than 40, exclusive of East Chatham-1974 previously mentioned, were: Central Essex-1973 (Got-2 and Dip-1); Harrow-1974 (Id-1); Stoney Point-1974 (Mod-1); and, Wheatley-1974 (Mod-1 and Dip-1).

In summary, there appear to be no geographic patterns of significant deviation from Hardy-Weinberg equilibrium for any system examined, although the populations of Central Essex and Stoney Point regions did show significant deviation from Hardy-Weinberg equilibrium for several loci and over two consecutive years. Also, departures from Hardy-Weinberg equilibrium in the pooled data of the entire southwestern Ontario population were significant for all loci studied, with the exception of Pgm-1 and Pgm-2 (Table 4).

Again, the significance of the Hardy-Weinberg equilibrium deviations, in each case, was due to a numerical deficiency of heterozygotes. Of the various explanations that could account for this deficiency, the most probable is population structuring. Individual populations sampled may be at

Table 4: Summary of chi-square analysis of deviations from Hardy-Weinberg equilibrium in the pooled sampling data at the loci found to be polymorphic in mouse populations of southwestern Ontario. The asterisks refer to populations which significantly differ from a Hardy-Weinberg equilibrium (* - P less than 0.05; ** - P less than 0.01).

	1973			1974		
ENZYME LOCUS	SAMPLE SIZE	CHI - SQUARE	P	SAMPLE SIZE	CHI- SQUARE	P
Id-1	262	19.0161	**	431	21.1224	**
Mod-1	282	101.8626	**	397	143.4432	**
Got-2	244	140.4004	**	467	20.9395	**
Dip-1	244	47.6384	**	467	347.3007	**
Gpd-1				458	57.7950	**
Pgm-1				467	0.0963	
Pgm-2				467	0.0477	

Hardy-Weinberg equilibrium, but when pooled show a significant deficiency in heterozygotic genotypes. This is the Wahlund effect.

D Genic Heterozygosity

A measure of genic variability, which can be applied to the study of natural populations, including those of the house mouse, is heterozygosity ($\underline{H}$). The overall amount of variation in a population may be estimated by the average frequency of heterozygotes per locus ($\overline{\underline{H}}$). This is simply obtained by averaging $\underline{H}$ over all loci sampled (Dobzhansky et al., 1977).

This survey of protein variation, as is generally the case, is not totally free of certain biases and limitations. To achieve unbiased estimates of genetic variation in natural populations several conditions should be met, including: large sample size; genomes tested directly from the natural population or removed from the original sample by only a few generations; a large sample of loci controlling diverse functions; and, a sample of loci unbiased by choice due to previously known variability (Lewontin, 1974).

In the present study, sample size for some regions was small, but pooled samples generally were large. The mice were wild caught. The loci chosen for the study were not randomly selected, since all had been previously studied and all controlled enzymes. The loci studied in 1974 animals were obviously biased since all loci found monomorphic in 1973 animals,

with the exception of Got-1, were eliminated from the survey. The overall heterozygosity in the 1974 study is less distorted, if those loci found monomorphic in 1973 are considered also monomorphic in 1974.

The data collected from the 1973 samples showed variation in four of the 9 loci examined. The average heterozygosity per individual per locus studied was 0.0941 (Table 5). For 1974 samples, because of the omission of most monomorphic loci, 8 of the 9 loci studied were variable. Only Got-1 was monomorphic. If the four loci found monomorphic for the 1973 animals were considered also monomorphic for the 1974 animals, overall estimate of heterozygosity was 0.0864 (Table 5).

A slight decrease in heterozygosity seemed to occur from 1973 to 1974 data based on all loci. When heterozygosity was determined for only polymorphic loci, the 1973 data proved to have a higher mean value, 0.2201, than the 1974, 0.1408 (Table 6). This may have been the result of the PGM-1 and PGM-2 data, which showed little variability. Upon elimination of these data, the estimate of heterozygosity based on the remaining 6 loci was still lower for the 1974 samples, 0.1816, than that for the 1973 samples. Greater heterozygosity was seen not only for the 1973 over the 1974 data for the pooled samples of southwestern Ontario, but also in several of the regional samples where comparison between 1973 and 1974 data was possible. This held true for all cases when 8 loci were considered and for three of 5 regional samples when 6 loci were considered for 1974 samples. Furthermore, this held true

Table 5: Heterozygosity at thirteen loci in regional populations of Mus musculus for 1973 and 1974. Ipo-1, Ldh-1, Trip-1, and Mpi-1 were considered monomorphic for 1974, since these loci were monomorphic for 1973 (an average of the 1974 sample size of all monitored loci for each region was used as the sample size for each of these loci).

		1973			1974		
REGION	LOCUS	SAMPLE SIZE	HETEROZYGOTE NUMBER	PROPORTION	SAMPLE SIZE	HETEROZYGOTE NUMBER	PROPORTION
Harrow	Id-1	16	2	0.1250	77	8	0.1039
	Mod-1	20	7	0.3500	68	1	0.0147
	Got-2	16	2	0.1250	91	23	0.2527
	Dip-1	16	3	0.1875	91	1	0.0110
	Dip-2				91	13	0.1429
	Gpd-1				91	22	0.2418
	Pgm-1				91	2	0.0220
	Pgm-2				91	3	0.0330
	Got-1	16	0	0	91	0	0
	Ipo-1	20	0	0	86	0	0
	Ldh-1	20	0	0	86	0	0
	Trip-1	20	0	0	86	0	0
	Mpi-1	20	0	0	86	0	0
MEAN HETEROZYGOSITY:							
per year				0.0853			0.0648
for both years	0.0674						

Table 5: continued

REGION	LOCUS	1973 SAMPLE SIZE	1973 HETEROZYGOTE NUMBER	1973 HETEROZYGOTE PROPORTION	1974 SAMPLE SIZE	1974 HETEROZYGOTE NUMBER	1974 HETEROZYGOTE PROPORTION
Sandwich West	Id-1				5	1	0.2000
	Mod-1				5	0	0
	Got-2				5	3	0.6000
	Dip-1				5	0	0
	Dip-2				5	0	0
	Gpd-1				5	2	0.4000
	Pgm-1				5	2	0.4000
	Pgm-2				5	0	0
	Got-1				5	0	0
	Ipo-1				5	0	0
	Ldh-1				5	0	0
	Trip-1				5	0	0
	Mpi-1				5	0	0
MEAN HETEROZYGOSITY: per year							0.1230

Table 5: continued

REGION	LOCUS	1973 SAMPLE SIZE	1973 HETEROZYGOTE NUMBER	1973 HETEROZYGOTE PROPORTION	1974 SAMPLE SIZE	1974 HETEROZYGOTE NUMBER	1974 HETEROZYGOTE PROPORTION
Central Essex	Id-1	48	19	0.3878	109	16	0.1468
	Mod-1	50	7	0.1400	97	8	0.0825
	Got-2	45	5	0.1111	114	50	0.4386
	Dip-1	45	13	0.2889	114	9	0.0790
	Dip-2				114	16	0.1404
	Gpd-1				110	33	0.3000
	Pgm-1				114	7	0.0614
	Pgm-2				114	0	0
	Got-1	45	0	0	114	0	0
	Ipo-1	50	0	0	111	0	0
	Ldh-1	50	0	0	111	0	0
	Trip-1	50	0	0	111	0	0
	Mpi-1	50	0	0	111	0	0
MEAN HETEROZYGOSITY:							
per year				0.1016			0.0962
for both years	0.0998						

Table 5: continued

REGION	LOCUS	1973 SAMPLE SIZE	1973 HETEROZYGOTE NUMBER	1973 HETEROZYGOTE PROPORTION	1974 SAMPLE SIZE	1974 HETEROZYGOTE NUMBER	1974 HETEROZYGOTE PROPORTION
Stoney Point	Id-1	157	59	0.3758	59	11	0.1864
	Mod-1	169	21	0.1243	62	4	0.0645
	Got-2	147	15	0.1020	69	16	0.2319
	Dip-1	147	45	0.3061	69	2	0.0290
	Dip-2				69	11	0.1594
	Gpd-1				68	17	0.2500
	Pgm-1				69	0	0
	Pgm-2				69	2	0.0290
	Got-1	147	0	0	69	0	0
	Ipo-1	170	0	0	67	0	0
	Ldh-1	170	0	0	67	0	0
	Trip-1	170	0	0	67	0	0
	Mpi-1	170	0	0	67	0	0
MEAN HETEROZYGOSITY:							
per year				0.0967			0.0723
for both years	0.0875						

Table 5: continued

REGION	LOCUS	1973 SAMPLE SIZE	1973 HETEROZYGOTE NUMBER	1973 HETEROZYGOTE PROPORTION	1974 SAMPLE SIZE	1974 HETEROZYGOTE NUMBER	1974 HETEROZYGOTE PROPORTION
Prairie Siding	Id-1	9	5	0.5556			
	Mod-1	10	4	0.4000			
	Got-2	8	1	0.1250			
	Dip-1	8	1	0.1250			
	Dip-2						
	Gpd-1						
	Pgm-1						
	Pgm-2						
	Got-1	8	0	0			
	Ipo-1	11	0	0			
	Ldh-1	11	0	0			
	Trip-1	11	0	0			
	Mpi-1	11	0	0			
MEAN HETEROZYGOSITY:							
per year				0.1264			

Table 5: continued

REGION	LOCUS	1973 SAMPLE SIZE	1973 HETEROZYGOTE NUMBER	1973 HETEROZYGOTE PROPORTION	1974 SAMPLE SIZE	1974 HETEROZYGOTE NUMBER	1974 HETEROZYGOTE PROPORTION
Paincourt	Id-1	16	1	0.0625	12	4	0.3333
	Mod-1	17	0	0	9	0	0
	Got-2	15	2	0.1333	15	3	0.2000
	Dip-1	15	3	0.2000	15	1	0.0667
	Dip-2				15	5	0.3333
	Gpd-1				15	4	0.2667
	Pgm-1				15	0	0
	Pgm-2				15	0	0
	Got-1	15	0	0	15	0	0
	Ipo-1	17	0	0	14	0	0
	Ldh-1	17	0	0	14	0	0
	Trip-1	17	0	0	14	0	0
	Mpi-1	17	0	0	14	0	0
MEAN HETEROZYGOSITY:							
per year				0.0410			0.0934
for both years	0.0666						

Table 5: continued

REGION	LOCUS	1973 SAMPLE SIZE	1973 HETEROZYGOTE NUMBER	1973 HETEROZYGOTE PROPORTION	1974 SAMPLE SIZE	1974 HETEROZYGOTE NUMBER	1974 HETEROZYGOTE PROPORTION
East Chatham	Id-1	15	4	0.2667	85	20	0.2353
	Mod-1	16	3	0.1875	78	8	0.1026
	Got-2	13	3	0.2308	87	38	0.4368
	Dip-1	13	2	0.1538	87	5	0.0575
	Dip-2				87	13	0.1494
	Gpd-1				84	36	0.4286
	Pgm-1				87	1	0.0115
	Pgm-2				87	0	0
	Got-1	13	0	0	87	0	0
	Ipo-1	16	0	0	85	0	0
	Ldh-1	16	0	0	85	0	0
	Trip-1	16	0	0	85	0	0
	Mpi-1	16	0	0	85	0	0
MEAN HETEROZYGOSITY:							
per year				0.0895			0.1573
for both years	0.1091						

Table 5: continued

REGION	LOCUS	1973 SAMPLE SIZE	1973 HETEROZYGOTE NUMBER	1973 HETEROZYGOTE PROPORTION	1974 SAMPLE SIZE	1974 HETEROZYGOTE NUMBER	1974 HETEROZYGOTE PROPORTION
Wheatley	Id-1				84	21	0.2500
	Mod-1				78	4	0.0513
	Got-2				86	30	0.3488
	Dip-1				86	6	0.0698
	Dip-2				86	5	0.0581
	Gpd-1				85	18	0.2118
	Pgm-1				86	0	0
	Pgm-2				86	5	0.0581
	Got-1				86	0	0
	Ipo-1				85	0	0
	Ldh-1				85	0	0
	Trip-1				85	0	0
	Mpi-1				85	0	0
MEAN HETEROZYGOSITY:							
per year							0.0779

Table 5: continued

MEAN HETEROZYGOSITY FOR POOLED REGIONS

YEAR	LOCI	SAMPLE SIZE	HETEROZYGOTE NUMBER	HETEROZYGOTE PROPORTION
1973	all 9 loci	2411	227	0.0941
1974	all 13 loci	5900	510	0.0864

Table 6: Heterozygosity at all polymorphic loci in regional populations of *Mus musculus*.

REGION	HETEROZYGOSITY (1973 animals based on 4 loci)	HETEROZYGOSITY (1974 animals based on 8 loci)	HETEROZYGOSITY (1974 animals based on 6 loci) *
Harrow	0.2000	0.1056	0.1335
Sandwich West		0.2000	0.2000
Central Essex	0.2340	0.1568	0.2006
Stoney Point	0.2258	0.1179	0.1540
Prairie Siding	0.1264		
Paincourt	0.0952	0.1531	0.2098
East Chatham	0.2105	0.1774	0.2362
Wheatley		0.1314	0.1663
Pooled for all regions	0.2201	0.1408	0.1816

Pooled for all regions for both years - 0.1584

* excluding Pgm-1 and Pgm-2

for three of 5 regional samples when heterozygosity was based on all loci (Table 5).

In the pooled data of the two years, heterozygosity at polymorphic loci and at all loci was 0.1584 (Table 6) and 0.0886 (Table 7), respectively. The value more indicative of the heterozygosity of the population is, of course, the one based on all loci examined and extrapolating from 1973 to 1974 populations for monomorphic loci. In this case, 8 of 13 loci were variable, with the average heterozygosity per individual being 0.0886 (Table 7).

This value falls well within the range of other investigations. Roderick et al. (1971) found feral populations of mice showed an average heterozygosity per population from 0.0760 to 0.1470 for 17 loci. Similar results were found by Selander et al. (1969) and Ruddle et al. (1969). Mus musculus is not unique in its genic variability. The average heterozygosity found per individual of the species Homo sapiens is 0.0740 (Harris, 1969) and 0.1230 for the species Drosophila pseudoobscura (Prakash et al., 1969).

Electrophoretic studies used to estimate genic heterozygosity detect only a proportion of the actual variation in protein structure since only about one-third of the amino acid substitutions result in change of the isoelectric point of polypeptides. Nevertheless, if the loci examined in this survey are representative of all loci, there must be thousands of polymorphisms per Mus population and each individual must be heterozygous for a significant portion of its genome.

Table 7: Heterozygosity at thirteen loci in the pooled samples of southwestern Ontario. The overall heterozygosity for 1973 and 1974 samples was calculated on the basis that loci monomorphic in 1973 were the same in 1974.

LOCUS	SAMPLE SIZE	HETEROZYGOTE NUMBER	PROPORTION
Id-1	692	171	0.2471
Mod-1	679	67	0.0986
Got-2	711	191	0.2686
Dip-1	711	91	0.1279
Dip-2	467	63	0.1349
Pgm-1	467	12	0.0256
Pgm-2	467	10	0.0214
Gpd-1	458	132	0.2882
Got-1	711	0	0
Ipo-1	737	0	0
Ldh-1	737	0	0
Trip-1	737	0	0
Mpi-1	737	0	0
Pooled	8311	737	0.0886

E Genetic Distance

Genetic distance is a measure of the genetic differences between populations as expressed by a function of gene frequencies (Nei, 1971; 1972). These genetic differences, which arise since a total population forming a species is generally not a random mating unit, are the result of the interaction of three possible factors: mutation, selection, and random genetic drift (Wright, 1943).

Malécot (1955) examined the correlation between geographic distance and genetic uniqueness of individual populations and found that individuals and populations living closeby tended to be more similar genetically than those living far apart. Similar conclusions were reached through a variety of measures of genetic distance proposed by a number of authors: Kimura and Weiss (1964); Cavalli-Sforza and Edwards (1967); Balakrishnan and Sanghvi (1968); and, Hedrick (1971). However, in many of these, it was not made clear what biological unit was being measured (Lewontin, 1974). From the genetic point of view, the most suitable measure of genetic distance appears to be the one proposed by Nei (1971; 1972), who developed a method by which the average number of codon differences per locus could be estimated from gene frequency data.

Nei (1971; 1972) proposed three different measures of genetic distance: the minimum, standard, and maximum estimates of detectable codon differences per locus. There is not much difference among these three measures when local

populations within one species are compared. In this case, therefore, any one is equally applicable.

In this study, standard genetic distance (D), between two populations, X and Y was used, which can be written as

$$D = D_{XY} - (D_X + D_Y) / 2$$

where

$$D_{XY} = -\log_e J_{XY}$$

$$D_X = -\log_e J_X$$

$$D_Y = -\log_e J_Y$$

with the probability of identity of two randomly chosen genes as $j_X = \sum x_i^2$ in population X, while it is $j_Y = \sum y_i^2$ in population Y. The frequencies of the i-th alleles in X and Y are x_i and y_i, respectively. The probability of identity of two genes, chosen at random, one from each of the two populations, is $j_{XY} = \sum x_i y_i$. J_X, J_Y, and J_{XY} are the designated arithmetic means of j_x, j_y, and j_{xy}, respectively, over all loci, including those monomorphic (Lewontin, 1974).

A few limitations of this method do exist. Since in the present study, only frequencies at highly polymorphic loci are used, and since as Nei (1971; 1972) points out, if

large numbers of loci are not used, genetic distances may deviate considerably from real values; only the relative values of the distances are useful.

Measurements of genetic distance were made from the 1974 mouse kidney enzyme data based upon 10 polymorphic loci (Id-1, Mod-1, Got-2, Dip-1, Peb-s, Pgm-1, Pgm-2, Dip-2, "Pgm-3", and, "Pgm-4") and 5 monomorphic loci (Got-1, Ipo-1, Ldh-1, Trip-1, and, Mpi-1). For these measurements, the 5 loci found monomorphic in 1973 animals were assumed to be so in 1974 animals. This assumption is supported by the Got-1 data for 1973 and 1974.

Table 8 displays the mean genetic distance based on all samples taken in southwestern Ontario. Visual examination of the data shows no obvious pattern, although the data suggest that there is as much genetic differentiation between populations of a single region as between populations of different regions. However, it is unknown whether these differences in genetic distance are significant.

Genetic distances based on overall regional allelic frequencies as opposed to individual populations are given in Table 9. This approach was used to minimize the effects of small sample size on genetic distance calculations, however, again, no obvious patterns or trends were observed.

According to previous studies, normally the closer geographically two regions are, the more similar genetically the inhabitants of those regions are expected to be (Nei, 1971; Nei and Imaizumi, 1966a, b). Whereas, there appear to be no

Table 8: Mean genetic distances for 1974 regional mouse populations based on samples from all sites.

	HARROW	SANDWICH WEST	CENTRAL ESSEX	STONEY POINT	PAINCOURT	EAST CHATHAM	WHEATLEY
WHEATLEY							0.0136
EAST CHATHAM						0.0236	0.0224
PAINCOURT					0.0425	0.0334	0.0340
STONEY POINT				0.0211	0.0399	0.0237	0.0172
CENTRAL ESSEX			0.0262	0.0226	0.0385	0.0242	0.0230
SANDWICH WEST		.....	0.0201	0.0162	0.0379	0.0196	0.0135
HARROW	0.0204	0.0171	0.0258	0.0201	0.0379	0.0272	0.0185

Table 9: Genetic distances for 1974 regional mouse populations based on regional allelic frequencies.

	HARROW	SANDWICH WEST	CENTRAL ESSEX	STONEY POINT	PAINCOURT	EAST CHATHAM	WHEATLEY
WHEATLEY							0
EAST CHATHAM						0	0.0049
PAINCOURT					0	0.0084	0.0160
STONEY POINT				0	0.0207	0.0066	0.0012
CENTRAL ESSEX			0	0.0049	0.0116	0.0009	0.0040
SANDWICH WEST		0	0.0083	0.0073	0.0269	0.0088	0.0084
HARROW	0	0.0091	0.0061	0.0011	0.0160	0.0068	0.0012

obvious trends in this direction, the coefficient of correlation (r) between geographic distance in miles and genetic distance for paired comparisons of 1974 samples from individual corn cribs gave an r value of 0.0779 (d.f. 594). This was found significant at the 5 percent level.

Figure 10 graphs a comparison of genetic distance versus geographic distance of Harrow versus all regions and Central Essex versus all regions. This graph illustrates that no obvious relationship between genetic and geographic distances is evident.

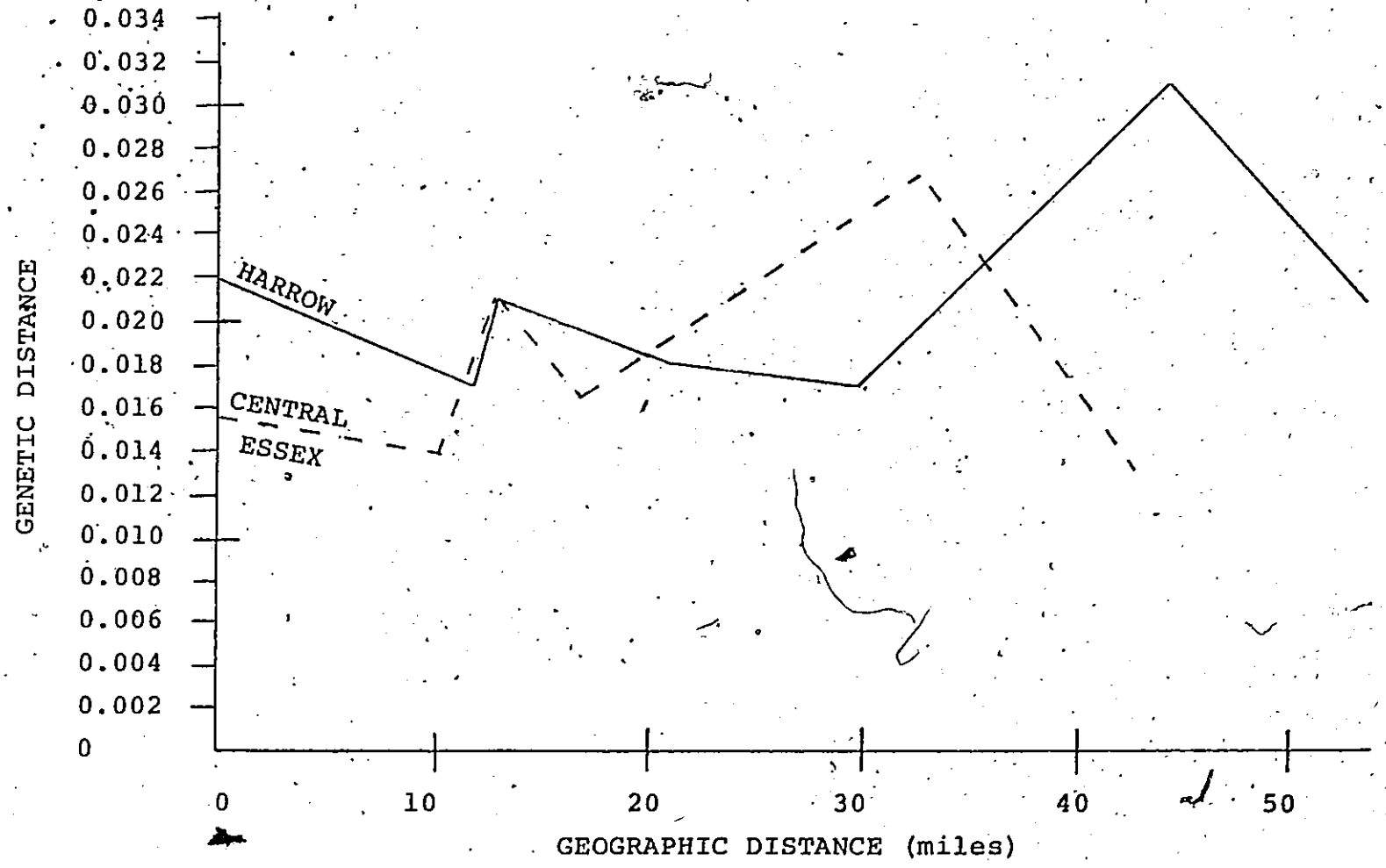
In summary, there appears to be only a weak correlation between geographic and genetic distances. This suggests that genetic drift within the populations, which should result in considerable differentiation, is being counteracted by another force or forces, perhaps selection and/or significant migration rate.

F Variance Analysis

Frequency variances were determined for the alleles at each locus as described by Cavalli-Sforza and Bodmer (1971). The observed variance has two components: sampling and genetic (Cavalli-Sforza and Bodmer, 1971). The former results from the entire population not being typed. The latter is the difference between the observed and sampling variance and reflects the unequal contribution of various genotypes to the next generation.

Three types of patterns may be observed among the

Figure 10: Genetic distance versus geographic distance for Harrow and Central Essex as compared to all other regions of southwestern Ontario.



different variances: sampling variance may equal observed variance; sampling variance may be greater than observed variance; or, sampling variance may be smaller than observed variance.

The first case, in which sampling variance equals observed variance could be explained by artificial subdivision of a panmictic population. In the second, where sampling variance is greater than observed variance, that is a frequency uniformity may be observed which is greater than expected for the sample sizes obtained then selection and/or a tendency for outbreeding may modulate the effect of genetic drift. Finally, if sampling variance is smaller than observed variance, frequency differentiation due to population subdivision may be observed. In other words, observed variance may be increased by population subdivision. However, on the other hand, observed variance may be decreased by migration between subpopulations and selection, resulting in sampling variance equalling observed variance.

Unfortunately, estimates of sampling variance are based on sampling from a large population and so are overestimates in the current study, since a large portion of the population, if not the entire population, is collected from any corn crib. Therefore, the genetic variance lies between the observed variance and an appropriately adjusted sampling variance.

Table 10 summarizes the allelic variances for each locus of populations sampled in both 1973 and 1974. Sample sizes from all regions monitored in 1973 were small, with the

Table 10: A summary of variances at seven loci controlling renal enzymes in 1973 and 1974 regional samples. (No. - sample size; σ_o - observed variance; σ_s - sampling variance; f - Wahlund's variance; * - to the -10).

		1973				1974						
REGION		Id-1	Mod-1	Got-2	Dip-1	Id-1	Mod-1	Got-2	Dip-1	Gpd-1	Pgm-1	Pgm-2
Harrow	No.	16	20	16	16	77	68	91	91	91	91	91
	σ_o	0.1125	0.0518	0.0156	0.0498	0.0031	0.0034	0.0022	0.0130	0.0105	0.0001	0.0027
	σ_s	0.0267	0.0310	0.0312	0.0458	0.0050	0.0060	0.0101	0.0127	0.0150	0.0003	0.0024
	f	0.4800	0.2364	0.1428	0.2464	0.0275	0.0555	0.0170	0.0561	0.0432	0.0066	0.1666
Sandwich West	No.	0	0	0	0	5	5	5	5	5	5	5
	σ_o					0	0	0	0	8.1633*	0	0
	σ_s					0.0180	0	0.0420	0.0320	0.0026	0.0320	0
	f					0		0	0	30.000*	0	
Central Essex	No.	49	50	45	45	109	97	114	114	110	114	114
	σ_o	0.0096	0.0168	0.0115	0.0178	0.0076	0.0057	0.0120	0.0037	0.0123	0.0013	0
	σ_s	0.0193	0.0146	0.0200	0.0204	0.0063	0.0078	0.0143	0.0047	0.0156	0.0026	0
	f	0.0386	0.0786	0.0496	0.0751	0.0923	0.0622	0.0487	0.0340	0.0549	0.0469	
Stoney Point	No.	157	169	147	147	59	62	69	69	68	69	69
	σ_o	0.0022	0.0019	0.0075	0.0099	0.0047	0.0049	0.0177	0.0136	0.0026	0	0.0001
	σ_s	0.0059	0.0045	0.0073	0.0063	0.0068	0.0046	0.0121	0.0091	0.0176	0	0.0003
	f	0.0100	0.0101	0.0307	0.0399	0.0563	0.0498	0.1100	0.0664	0.0113		0.0083
Prairie Siding	No.	9	10	8	8	0	0	0	0	0	0	0
	σ_o	1.2346*	0	0	0.0131							
	σ_s	0.0264	0.0240	0.0307	0.0299							
	f	5.1948*	0	0	0.0610							
Paincourt	No.	16	17	15	15	12	9	15	15	15	15	15
	σ_o	0.0275	0	0.0273	0.0803	0.0534	0	0.0197	0.0397	0.0016	0	0
	σ_s	0.0303	0	0.0340	0.0276	0.0431	0	0.0464	0.0298	0.0495	0	0
	f	0.1220		0.1395	0.1270	0.2406		0.0804	0.1712	0.0066		
East Chatham	No.	15	16	13	13	85	78	87	87	84	87	87
	σ_o	0.0122	0.0019	0.0171	0.0062	0.0089	0.0053	0.0067	0.0359	0.0038	0.0003	0
	σ_s	0.0285	0.0035	0.0333	0.0516	0.0141	0.0091	0.0214	0.0113	0.0232	0.0007	0
	f	0.0550	0.0228	0.0759	0.0295	0.0586	0.0654	0.0269	0.2585	0.0162	0.0666	
Wheatley	No.	0	0	0	0	84	78	86	86	85	86	86
	σ_o					0.0049	0.0033	0.0027	0.0348	0.0011	0	0.0004
	σ_s					0.0063	0.0051	0.0074	0.0082	0.0091	0	0.0009
	f					0.0346	0.0475	0.0152	0.1617	0.0053		0.0162

exception of Stoney Point, and smaller, for the most part than in 1974. Consequently, much of the observed variance between subpopulations of the regions could be attributed to sampling variance. In fact, the difference between sampling and observed variances proved, in many cases, to be negative.

When the F test (Sokal and Rohlf, 1969) was applied to determine whether the observed variance was significantly different from the sampling variance for all regional and pooled calculations (Table 11), only two F values were significant. These involved the Dip-1 locus in two samples collected in 1974. Since most F values were not significant, there was not an appreciable difference between the observed and sampling variances. As a result, in kidney enzyme loci studied, sampling variance could account for most of the observed variance and, therefore, necessitating little contribution from genetic variance. This conclusion must be tempered with the realization that most sample sizes were quite small, an average of about 12 in each of the years.

Therefore, sampling variance could account for all of the observed variance, provided that sampling variance is based on sampling from a population approaching infinite size. Because the populations being sampled are much smaller, the sampling variance is considerably less than the maximum seen in Table 10.

Table 12 presents the variances calculated from the pooled samples for the various loci of concern. In most cases, the sampling variance is smaller than the observed

Table 11: F values for $\sigma o/\sigma s$ for seven loci controlling renal enzymes in 1973 and 1974 regional and pooled samples. The asterisks refer to the level of significance of the F values (* - 5%; ** - 1%).

1973

REGION	Id-1	Mod-1	Got-2	Dip-1
Harrow	4.2134	1.6709	0.5000	1.0873
Sandwich West				
Central Essex	0.4974	1.1506	0.5750	0.8725
Stoney Point	0.3728	0.4222	1.0273	1.5714
Prairie Siding	0	0	0	0.4381
Paincourt	0.9075	0	0.8029	2.9094
East Chatham	0.4280	0.5428	0.5135	0.1201
Wheatley				
Pooled (based on Table 11)	1.5328	1.5963	1.1666	1.4276

1974

REGION	Id-1	Mod-1	Got-2	Dip-1	Gpd-1	Pgm-1	Pgm-2
Harrow	0.6200	0.5666	0.2178	1.1236	0.7000	0.3333	1.1250
Sandwich West	0	0	0	0	0	0	0
Central Essex	1.2063	0.7307	0.8391	0.7872	0.7884	0.5000	0
Stoney Point	0.6911	1.0652	1.4628	1.4945	0.1477	0	0.3333
Prairie Siding							
Paincourt	1.2389	0	0.4245	1.3322	0.0323	0	0
East Chatham	0.6312	0.5824	0.3130	3.1769*	0.1637	0.4285	0
Wheatley	0.7777	0.6470	0.3648	4.2439**	0.1208	0	0.4444
Pooled (based on Table 11)	1.0752	0.7727	2.0413	2.9042	0.4775	0.8181	1.0000

Table 12: A summary of variances at seven loci controlling renal enzymes in the pooled samples of 1973 and 1974. (σ_o - observed variance; σ_s - sampling variance).

1973	Id-1	Mod-1	Got-2	Dip-1
σ_o	0.0233	0.0174	0.0175	0.0227
σ_s	0.0152	0.0109	0.0150	0.0159

1974	Id-1	Mod-1	Got-2	Dip-1	Gpd-1	Pgm-1	Pgm-2
σ_o	0.0100	0.0051	0.0296	0.0273	0.0085	0.0009	0.0007
σ_s	0.0093	0.0066	0.0145	0.0094	0.0178	0.0011	0.0007

variance.

Therefore, since sampling and observed variances are not significantly different, the population under consideration may be panmictic. However, another possibility is that the observed variance was being decreased by migration and/or selection, with the population actually subdivided. Analysis of another measurement of variance, Wahlund's variance, allows further conclusions to be drawn on population structure.

G Wahlund's Variance

Populations of plants, animals, and humans are structured (Cavalli-Sforza, 1959; 1963; Nei and Imaizumi, 1966a, b). The actual structure of a population may be the result of different characterisitcs such as size, mobility, and mating pattern. Furthermore, an overall population may be divided into subpopulations or isolates as described by Wahlund in 1928 or neighbourhoods as discussed by Wright in 1943 (Cavalli-Sforza and Bodmer, 1971). In structured populations, the gene frequencies may diverge simply by chance if the subpopulations are small (Wright, 1943) or because different environments favour different genotypes. Wahlund's variance is a measure of population subdivision through gene frequency differentiation (Cavalli-Sforza and Bodmer, 1971). This measure is calculated from

$$f = \frac{\sigma^2}{\bar{p}\,\bar{q}}$$

where f is Wahlund's coefficient, σ^2 is genetic variance. $\bar{p}$ and $\bar{q}$ are mean pooled allelic frequencies in the population under consideration.

Hardy-Weinberg equilibrium deviations (Table 3) as well as ecological data suggest that the populations of Mus under consideration are subdivided. Therefore, use of Wahlund's variance is justified.

Wahlund's coefficients based on each locus studied are presented in Table 10 for populations of each region. In this instance, Wahlund's variance is based upon observed variance, which assumes that each population sampled is complete, that is, there is no sampling variance. Since this is not always the case, this calculated Wahlund's variance, therefore, is probably a slight overestimate of the actual variance. However, Wahlund's variance based on genetic variance in most cases would be a "gross" underestimate since genetic variance, as pointed out previously, is itself an underestimate.

All regions with an appreciable sample size displayed heterogeneity for Wahlund's variance at all loci studied except Pgm-1 and Pgm-2. These two loci, because they were predominantly represented by only one allele, were characterized by a negligible Wahlund's variance in many regions.

The Wahlund's variance ranged from zero to 0.4800 and to 0.2585 for 1973 and 1974 populations, respectively. The mean of the Wahlund's variances is presented in Table 13. The Harrow-1973 population had the highest mean value, 0.2764, and Paincourt-1973, the lowest, 0.0152. The mean value of

Table 13: A summary of mean values of Wahlund's variance for 1973 and 1974 regional and pooled samples.

REGION	1973	1974	1973/1974
Harrow	0.2764	0.0532	0.1343
Central Essex	0.0604	0.0484	0.0528
Stoney Point	0.0226	0.0431	0.0357
Prairie Siding	0.0152		
Paincourt	0.1471	0.0712	0.0599
East Chatham	0.0458	0.0703	0.0614
Wheatley		0.0400	
TOTAL	0.0946	0.0466	0.0624

Wahlund's variance based on genetic variance derived directly from the observed variance and the lower limit based on the genetic variance derived from the difference between the observed and sampling variances of the pooled samples are presented in Table 14. The mean value of the upper limit of Wahlund's variance was 0.0897 and 0.0870 for 1973 and 1974 samples, respectively; the mean value of the lower limit of Wahlund's variance was 0.0264 and 0.0500 for 1973 and 1974 samples, respectively. The mean values for both years were 0.0880 (upper limit) and 0.0382 (lower limit).

Because ecological data and observations involving Hardy-Weinberg equilibrium suggest population subdivision, a Wahlund's variance can be calculated and used in other population analysis.

H Wahlund's Variance and Migration, Population Size

The overall Mus musculus population of southwestern Ontario appears to be subdivided. Each of the subpopulations may very well be drifting randomly, which would suggest a pattern of greater variance between populations separated by greater geographic distance be present. Also, the greater the number of subpopulations being compared, the greater the value of Wahlund's variance for the complete sample should be. However, this type of pattern would only be expected if there were no other factors counteracting random genetic drift.

To get a better grasp of the relationship between Wahlund's variance and migration and population size, 1974

Table 14: Upper and lower limits of Wahlund's variance for the pooled samples of southwestern Ontario.

1973	Id-1	Mod-1	Got-2	Dip-1	Mean
Upper limit	0.0993	0.0934	0.0738	0.0924	0.0897
Lower limit	0.0344	0.0343	0.0100	0.0272	0.0264

1974	Id-1	Mod-1	Got-2	Dip-1	Gpd-1	Pgm-1	Pgm-2	Mean
Upper limit	0.0835	0.0647	0.1340	0.1479	0.0366	0.0713	0.0712	0.0870
Lower limit	0.0324		0.0677	0.0953			0.0047	0.0500

1973/1974	Mean
Upper limit	0.0880
Lower limit	0.0382

samples from three regions, where sample size exceeds 50, were studied: Harrow, Stoney Point, and East Chatham. This involves comparing:

(a) Wahlund's variance based on three samples pooled,
(b) Wahlund's variance based on samples from two regions at a time, and
(c) Wahlund's variance based on samples from each region.

Table 15 presents the variance measurements for each of the three regions paired and for all three regions pooled. Figure 11 illustrates the relationship between geographic distance and the upper limit of Wahlund's variance for 5 loci for the three comparisons discussed above.

If the effect of genetic drift were not being offset by any other pressures, the type of pattern that would be expected for this kind of comparison would be the highest value of Wahlund's variance when all three regions are considered (a), next highest for any two regions separated by a greatest geographical distance (b), and finally, the lowest variance should be seen within each individual region (c). As is evident from Figure 11, this pattern did not occur consistently for any of the loci.

Therefore, other moderating pressures must be counteracting the effects of genetic drift. These could be mutation, migration, and natural selection. It is however, unlikely that the mutation rate would be high enough to affect genetic

Table 15: Variance comparisons among three regions for 1974 animals. (σo - observed variance; σs - sampling variance; f - Wahlund's variance, upper limit).

REGIONS		Id-1	Mod-1	Got-2	Dip-1	Gpd-1	Pgm-1	Pgm-2
Harrow - East Chatham	σo	0.0073	0.0068	0.0344	0.0302	0.0077	0.0002	0.0014
	σs	0.0103	0.0093	0.0156	0.0108	0.0188	0.0005	0.0012
	f	0.0546	0.0939	0.1561	0.1536	0.0322	0.0274	0.1741
Harrow - Stoney Point	σo	0.0045	0.0047	0.0094	0.0135	0.0079	0.00007	0.0015
	σs	0.0064	0.0053	0.0109	0.0101	0.0161	0.0002	0.0015
	f	0.0450	0.0585	0.0644	0.0606	0.0327	0.0115	0.1035
East Chatham - Stoney Point	σo	0.0094	0.0056	0.0403	0.0251	0.0033	0.0002	0.00009
	σs	0.0111	0.0077	0.0173	0.0089	0.0206	0.0004	0.0001
	f	0.0742	0.0629	0.1947	0.1458	0.0143	0.0685	0.0143
Harrow - East Chatham - Stoney Point	σo	0.0075	0.0051	0.0329	0.0257	0.0069	0.0001	0.0010
	σs	0.0094	0.0071	0.0146	0.0103	0.0185	0.0004	0.0010
	f	0.0617	0.0640	0.1590	0.1288	0.0288	0.0295	0.1086

Figure 11: An illustration of the relationship between geographic distance and Wahlund's variance at three levels: regions; pairs of regions; and, three regions pooled. Migration rates between regions are included. (f - Wahlund's variance; m - migration rate).

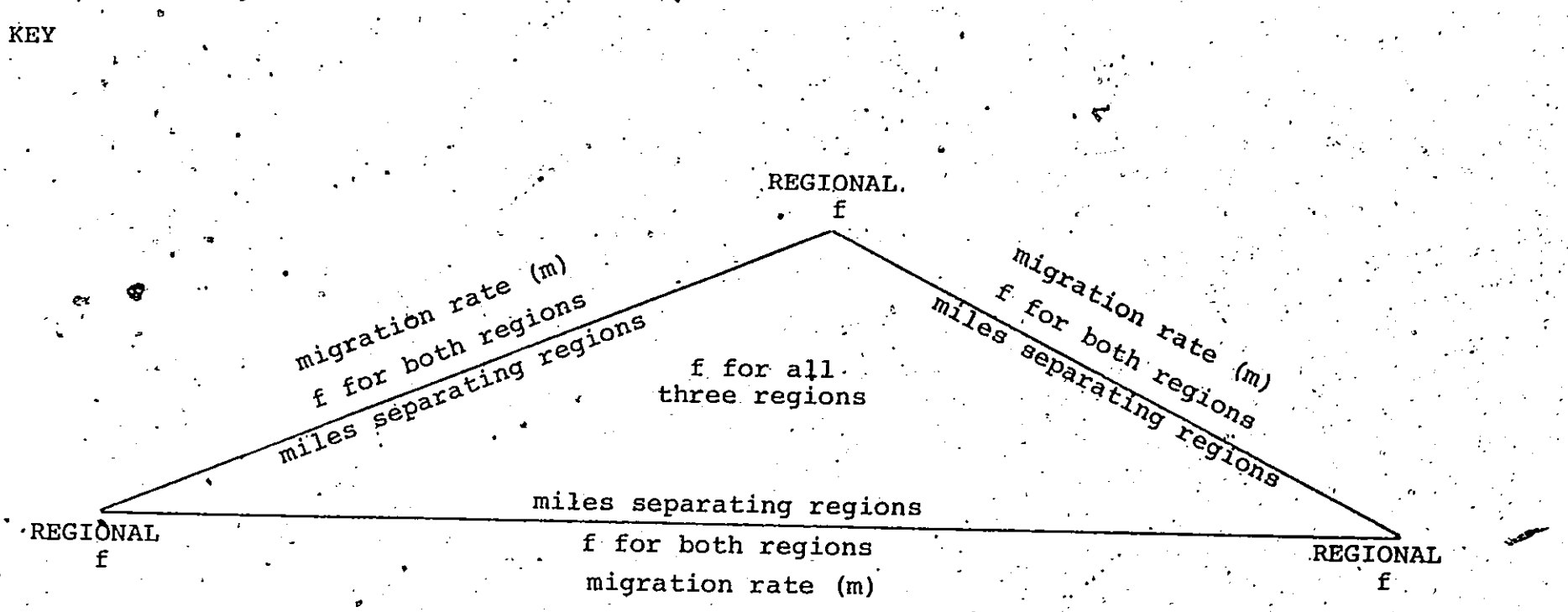
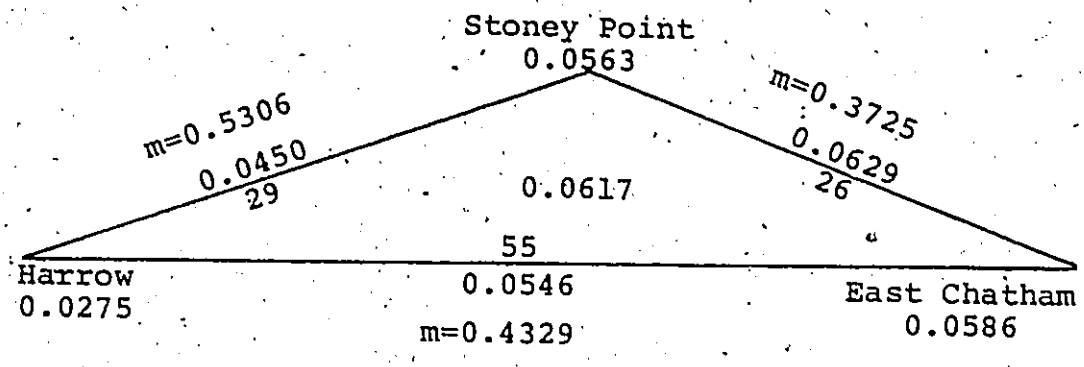


Figure 11: continued

Id-1



Mod-1

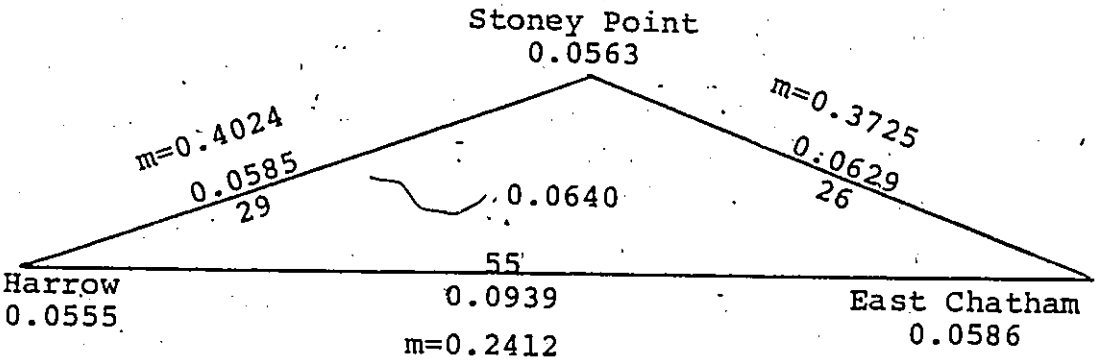
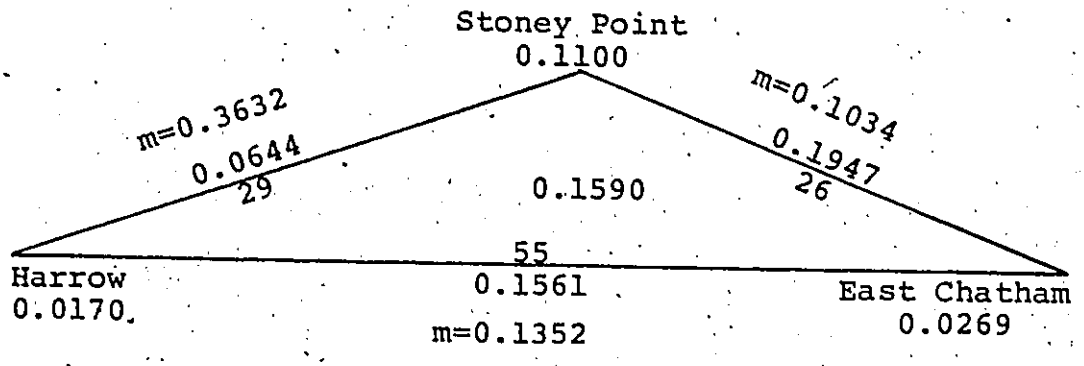


Figure 11: continued

Got-2



Dip-1

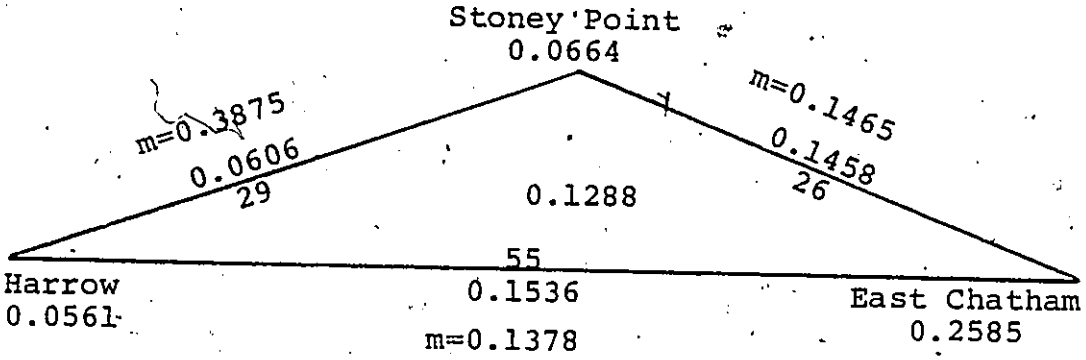
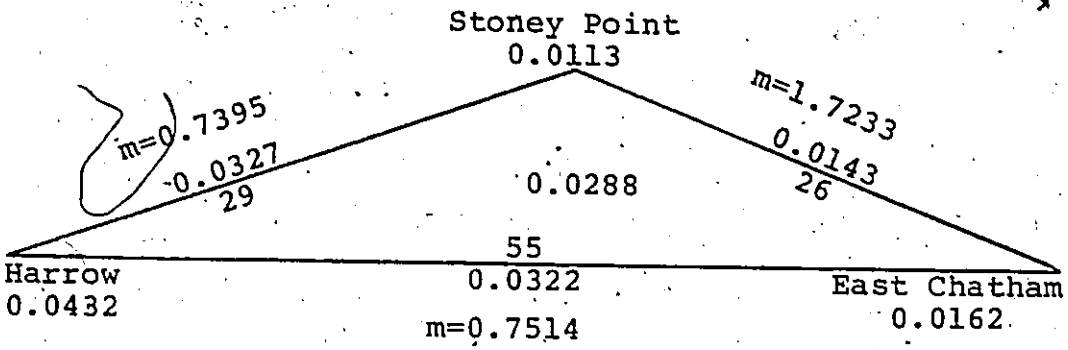


Figure 11: continued

Gpd-1



drift, and this, therefore, leaves either migration, selection, or a combination of both as the opposing pressures.

The simplest hypothesis involves migration. Migration rate estimates for the entire population for each locus are presented in Table 16. The formula (Cavalli-Sforza and Bodmer, 1971) used was

$$m = \frac{1}{4N}\left(\frac{1}{f} - 1\right)$$

which is a rearrangement of the formula for Wahlund's variance written as

$$f = \frac{\sigma^2}{\bar{p}\,\bar{q}} = \frac{1}{1 + 4Nm}$$

with σ^2 as genetic variance, $\bar{p}$ and $\bar{q}$ as the mean allelic frequencies, N as the genetically effective population size and m as the migration rate.

The value of N for the purposes of migration rate calculations was taken to be either 10 or 20. These are approximations of the lower and upper limits of the effective population size based on founder populations sizes, as determined by investigations of populations from these regions (Hawkeswood, 1975; Topping, 1975). The upper limits of Wahlund's variance were used.

In each case, the migration rate given is that which is required to counteract the effects of random genetic drift to maintain the appropriate value of Wahlund's variance,

Table 16: Migration rates based on observed variances for pooled samples of southwestern Ontario.

YEAR	EFFECTIVE POPULATION SIZE	Id-1	Mod-1	Got-2	Dip-1	Gpd-1	Pgm-1	Pgm-2
1973	10	0.2267	0.2426	0.3137	0.2455			
	20	0.1133	0.1213	0.1568	0.1227			
1974	10	0.2744	0.3613	0.1615	0.1440	0.6580	0.3256	0.3261
	20	0.1372	0.1806	0.0807	0.0720	0.3290	0.1628	0.1630
1973/1974 pooled mean								
	10	0.2505	0.3019	0.2376	0.1947			
	20	0.1252	0.1509	0.1187	0.0973			

1973/1974 mean for all loci

10	0.2981
20	0.1490

in the above, if no other pressures are acting.

If the migration rate is small enough, then no other pressures need to be invoked to account for the Wahlund's variance of the population. If the migration rate is very large and therefore, unlikely, then another explanation must be sought. That is, if migration rate is high, then another factor like natural selection as a buffer to random genetic drift must be invoked as explanation. Of course, both migration and selection may be in operation together.

Based on the upper limit of Wahlund's variance, it appears that migration alone is enough to effectively oppose genetic drift at either founder population size for all loci but one, Gpd-1 (N=10, 1974). The mean estimates of the migration rate for all loci under consideration would be 0.2981 (N=10) and 0.1490 (N=20).

If Wahlund's variance is close to its upper limit and the effective population size falls somewhere between the lower and upper limits specified, then migration alone may well counteract random genetic drift.

I Additional Loci Studied

In addition to the loci already discussed, two others were examined in the mouse kidney samples: PEP-S and DIP-2. These loci were not pooled with the rest because there was some question as to the consistency in interpretting the electrophoretic results because of band smearing.

PEP-S is described in the human by Rapley et al. (1971).

and in the mouse by Nichols and Ruddle (1973). The banding patterns have been previously described (see Methods) and are illustrated in Figure 5. Table 17 presents the number of animals for each of the phenotypes from the population studied. PEP-S slow (S) occurred more frequently than the other two phenotypes in 1973 samples and PEP-S fast (F), more frequently in those of 1974. There was an apparent deficiency of heterozygotes; the system was not pursued further.

DIP-2 patterns have been described previously (see Methods) and are illustrated in Figure 5. Table 18 presents the data for DIP-2; the fast form of DIP-2 is most common in the populations studied.

Table 17: A summary of phenotypes of Peptidase-S in Mus musculus populations. (S - slow; F - fast).

REGION	1973 SAMPLE SIZE	1973 S	1973 S/F	1973 F	1974 SAMPLE SIZE	1974 S	1974 S/F	1974 F
Harrow	16	8	1	7	91	42	0	49
Sandwich West					5	1	0	4
Central Essex	45	28	1	16	114	22	0	92
Stoney Point	147	97	4	46	69	21	0	48
Prairie Siding	8	4	0	4				–
Paincourt	15	8	0	7	15	6	0	9
East Chatham	13	9	2	2	87	24	1	62
Wheatley					86	32	0	54
Pooled for all regions	244	154	8	82	467	148	1	318

Table 18: A summary of phenotypes of Dipeptidase-2 in 1974 *Mus musculus* populations. (S - slow; F - fast).

REGION	SAMPLE SIZE	S	S/F	F
Harrow	91	4	13	74
Sandwich West	5	1	0	4
Central Essex	114	16	16	82
Stoney Point	69	2	11	56
Paincourt	15	1	5	9
East Chatham	87	10	13	64
Wheatley	86	13	5	68
Pooled for all regions	467	47	63	357

IV ERYTHROCYTIC PHOSPHOGLUCOMUTASE STUDY

A The Rare Alleles

In addition to the PGM kidney enzyme of the 1974 mice previously discussed, the phosphoglucomutases in the erythrocytes of samples from 1974, 1975, and 1976 were studied. Electrophoretic banding patterns proved to be similar for the blood and kidney enzymes for each of the 467 mice from 1974 screened and compared. Therefore, the same loci control the blood and kidney PGM-1 and PGM-2.

Pgm-1^e, Pgm-2^b, and Pgm-2^c alleles were found at very low frequency, and therefore, these two loci were especially informative in studies of the mouse populations of southwestern Ontario.

The frequencies of these rare alleles for erythrocytic PGM are presented in Tables 19 and 20.

Examination of the distribution of the rare alleles in only the regions made up of three or more populations, revealed one of four patterns: the alleles widely present throughout the region; the alleles present only in contiguous farms; the alleles present in only one population of a region; and, the alleles totally absent from the populations of a particular region.

Furthermore, analysis of the data over three consecutive years showed the occurrence of the rare alleles in the mouse

Table 19: A summary of PGM-1 phenotypes and allelic frequencies of Pgm-1^a. The asterisks refer to populations which significantly differ from a Hardy-Weinberg equilibrium (* = probability less than 0.05). (SS = sample size).

SITE / REGION	1974 SS	1974 PGM-1 PHENOTYPES AA	AE	EE	1974 Pgm-1^a FREQUENCY	1975 SS	1975 PGM-1 PHENOTYPES AA	AE	EE	1975 Pgm-1^a FREQUENCY	1976 SS	1976 PGM-1 PHENOTYPES AA	AE	EE	1976 Pgm-1^a FREQUENCY
Bondy (crib)	67	64	3	0	0.0224						42	42	0	0	0
Bondy (barn)	10	10	0	0	0	30	23	7	0	0.1167	74	56	18	0	0.1216
K.P. Martin						19	17	2	0	0.0526	64	64	0	0	0
D. Martin (crib)	36	34	2	0	0.0278	29	26	3	0	0.0517	38	37	1	0	0.0132
D. Martin (barn)						34	23	10	1	0.1765					
Pidgeon	19	18	1	0	0.0263	59	59	0	0	0	11	9	2	0	0.0909
Richardson	13	13	0	0	0										
Langois						17	16	1	0	0.0294					
Bennett	25	25	0	0	0										
Parkes East	25	0	0	0	0						50	33	15	2	0.1900
Parkes West											19	19	0	0	0
HARROW	195	189	6	0	0.0154	188	164	23	1	0.0665	298	260	36	2	0.0671
Walters	9	7	2	0	0.1111										
SANDWICH WEST	9	7	2	0	0.1111										
Chapo	8	7	1	0	0.0625										
Rocheleau East	34	34	0	0	0	90	90	0	0	0					
Rocheleau West	15	14	1	0	0.0333										
McKim	19	19	0	0	0										
Maitre North	68	64	4	0	0.0294	94	75	17	2	0.1117	123	116	7	0	0.0285
Maitre South	30	27	3	0	0.0500										
Price North	22	19	3	0	0.0682						27	27	0	0	0
Price South	17	15	2	0	0.0588						25	25	0	0	0
CENTRAL ESSEX	213	199	14	0	0.0329	184	165	17	2	0.0571	175	168	7	0	0.0256
E. Comartin	17	17	0	0	0						157	157	0	0	0
L. Comartin	19	19	0	0	0										
Houle	120	120	0	0	0	49	49	0	0	0	200	200	0	0	0
Baillargeon	13	13	0	0	0	25	24	1	0	0.0200	7	7	0	0	0
Nussey	37	37	0	0	0	12	12	0	0	0					
STONEY POINT	206	206	0	0	0	86	85	1	0	0.0058	364	364	0	0	0
Caron											38	38	0	0	0
Coulet											31	31	0	0	0
PRAIRIE SIDING											69	69	0	0	0
Belanger	25	24	1	0	0.0200	15	15	0	0	0	41	41	0	0	0
G. Belanger	13	13	0	0	0	44	43	1	0	0.0114	44	43	1	0	0.0114
Faubert	10	10	0	0	0	29	29	0	0	0					
Roy III											48	48	0	0	0
Chenick						35	35	0	0	0					
Roy XII											39	39	0	0	0
Ouellette											51	51	0	0	0
PAINCOURT	48	47	1	0	0.0104	123	122	1	0	0.0041	223	222	1	0	0.0022
Communication Road	22	22	0	0	0										
Snobdelen	18	18	0	0	0										
Nash Gravel Pit	8	8	0	0	0										
Nash	7	6	1	0	0.0714										
Brown	27	27	0	0	0										
Thatcher	4	4	0	0	0										
Johnston	17	17	0	0	0										
Lenover	24	24	0	0	0										
EAST CHATHAM	127	126	1	0	0.0039										
C. Wilkinson	55	55	0	0	0						28	28	0	0	0
G. Wilkinson	1	1	0	0	0										
N.R. Wilkinson	14	14	0	0	0	13	13	0	0	0					
James	37	37	0	0	0										
Leslie	55	54	1	0	0.0091										
WHEATLEY	162	161	1	0	0.0031	13	13	0	0	0	28	28	0	0	0
Pooled for all regions	960	935	25	0	0.0130	594	549	42	3	0.0404*	1157	1111	44	2	0.0208*

Table 20: A summary of PGM-2 phenotypes and allelic frequencies of Pgm-2[b] and Pgm-2[c]. The asterisks refer to populations which significantly differ from a Hardy-Weinberg equilibrium (** - probability less than 0.01). (SS - sample size; FREQU. - frequency).

1974

SITE / REGION	SS	PGM-2 PHENOTYPES AA	AB	BB	BC	CC	AC	Pgm-2[b] FREQU.	Pgm-2[c] FREQU.
Bondy (crib)	67	66	1	0	0	0	0	0.0075	0
Bondy (barn)	10	10	0	0	0	0	0	0	0
D. Martin	36	36	0	0	0	0	0	0	0
Pidgeon	19	19	0	0	0	0	0	0	0
Richardson	13	13	0	0	0	0	0	0	0
Bennett	25	22	3	0	0	0	0	0.0600	0
Parkes East	25	25	0	0	0	0	0	0	0
HARROW	195	191	4	0	0	0	0	0.0103	0
Walters	9	9	0	0	0	0	0	0	0
SANDWICH WEST	9	9	0	0	0	0	0	0	0
Chapo	8	8	0	0	0	0	0	0	0
Rocheleau East	34	34	0	0	0	0	0	0	0
Rocheleau West	15	15	0	0	0	0	0	0	0
McKim	19	19	0	0	0	0	0	0	0
Maitre North	68	68	0	0	0	0	0	0	0
Maitre South	30	30	0	0	0	0	0	0	0
Price North	22	21	1	0	0	0	0	0.0227	0
Price South	17	17	0	0	0	0	0	0	0
CENTRAL ESSEX	213	212	1	0	0	0	0	0.0023	0
E. Comartin	17	17	0	0	0	0	0	0	0
L. Comartin	19		19	0	0	0	0	0	0
Houle	120	109	9	2	0	0	0	0.0542	0
Baillargeon	13	13	0	0	0	0	0	0	0
Nussey	37	37	0	0	0	0	0	0	0
STONEY POINT	206	195	9	2	0	0	0	0.0316	0
Belanger	25	25	0	0	0	0	0	0	0
G. Belanger	13	13	0	0	0	0	0	0	0
Faubert	10	10	0	0	0	0	0	0	0
PAINCOURT	48	48	0	0	0	0	0	0	0
Communication	22	22	0	0	0	0	0	0	0
Snobdelen	18	18	0	0	0	0	0	0	0
Nash Gravel Pit	8	8	0	0	0	0	0	0	0
Nash	7	7	0	0	0	0	0	0	0
Brown	27	27	0	0	0	0	0	0	0
Thatcher	4	4	0	0	0	0	0	0	0
Johnston	17	17	0	0	0	0	0	0	0
Lenover	24	24	0	0	0	0	0	0	0
EAST CHATHAM	127	127	0	0	0	0	0	0	0
C. Wilkinson	55	52	3	0	0	0	0	0.0273	0
G. Wilkinson	1	1	0	0	0	0	0	0	0
N.R. Wilkinson	14	14	0	0	0	0	0	0	0
James	37	34	3	0	0	0	0	0.0404	0
Leslie	55	55	0	0	0	0	0	0	0
WHEATLEY	162	156	6	0	0	0	0	0.0185	0
Pooled for all regions **	960	938	20	2	0	0	0	0.0125	0

Table 20: continued

1975

SITE / REGION	SS	PGM-2 PHENOTYPES AA	AB	BB	BC	CC	AC	Pgm-2[b] FREQU.	Pgm-2[c] FREQU.
Bondy (barn)	30	28	2	0	0	0	0	0.0333	0
K.P. Martin	19	19	0	0	0	0	0	0	0
D. Martin (crib)	29	28	1	0	0	0	0	0.0172	0
D. Martin (barn)	34	28	6	0	0	0	0	0.0882	0
Pidgeon	59	50	8	0	0	0	1	0.0678	0.0085
Langois	17	17	0	0	0	0	0	0	0
HARROW	188	170	17	0	0	0	1	0.0452	0.0027
Rocheleau East	90	90	0	0	0	0	0	0	0
Maitre North	94	91	3	0	0	0	0	0.0160	0
CENTRAL ESSEX	184	181	3	0	0	0	0	0.0082	0
Houle	49	46	2	0	0	0	1	0.0204	0.0102
Baillargeon	25	25	0	0	0	0	0	0	0
Nussey	12	12	0	0	0	0	0	0	0
STONEY POINT	86	83	2	0	0	0	1	0.0116	0.0058
Belanger	15	15	0	0	0	0	0	0	0
G. Belanger	44	43	0	0	0	0	1	0	0.0114
Faubert	29	29	0	0	0	0	0	0	0
Chenick	35	33	2	0	0	0	0	0.0286	0
PAINCOURT	123	120	2	0	0	0	1	0.0081	0.0041
N.R. Wilkinson	13	12	0	0	0	1	0	0	0.0769
WHEATLEY	13	12	0	0	0	1	0	0	0.0769
Pooled for all regions **	*594	566	24	0	0	1	3	0.0202	0.0042

1976

Bondy (crib)	42	42	0	0	0	0	0	0	0
Bondy (barn)	74	66	6	1	0	0	1	0.0541	0.0067
K.P. Martin	64	64	0	0	0	0	0	0	0
D. Martin (crib)	38	38	0	0	0	0	0	0	0
Pidgeon	11	8	0	3	0	0	0	0.2727	0
Parkes East	50	49	1	0	0	0	0	0.0100	0
Parkes West	19	15	4	0	0	0	0	0.1053	0
HARROW	298	282	11	4	0	0	1	0.0319	0.0017
Maitre North	123	123	0	0	0	0	0	0	0
Price North	27	13	0	0	0	3	11	0	0.3148
Price South	25	18	1	1	0	1	4	0.0600	0.1200
CENTRAL ESSEX	175	154	1	1	0	4	15	0.0086	0.0657
E. Comartin	157	157	0	0	0	0	0	0	0
Houle	200	194	6	0	0	0	0	0.0150	0
Baillargeon	7	7	0	0	0	0	0	0	0
STONEY POINT	364	358	6	0	0	0	0	0.0082	0
Caron	38	38	0	0	0	0	0	0	0
Coulet	31	31	0	0	0	0	0	0	0
PRAIRIE SIDING	69	69	0	0	0	0	0	0	0
Belanger	41	41	0	0	0	0	0	0	0
G. Belanger	44	44	0	0	0	0	0	0	0
Roy III	48	48	0	0	0	0	0	0	0
Roy XII	39	38	1	0	0	0	0	0.0128	0
Ouellette	51	51	0	0	0	0	0	0	0
PAINCOURT	223	222	1	0	0	0	0	0.0022	0
C. Wilkinson	28	25	3	0	0	0	0	0.0536	0
WHEATLEY	28	25	3	0	0	0	0	0.0536	0
Pooled for all regions **	1157	1110	22	5	0	4	16	0.0138	0.0104

population of any of the corn cribs fits one of the following three patterns: the alleles persist over at least two years; the alleles vacillate between being present or absent in a particular population; and, the alleles are not detected.

Fluctuations in allelic frequency for all three rare alleles occurred from year to year in any one population and in the pooled data suggesting random genetic drift was in effect.

B The $Pgm\text{-}1^{e}$ Allele

The frequency of $Pgm\text{-}1^{e}$ ranged from zero to 0.1900 in the populations over the three years, with the highest frequency occurring in the pooled data for 1975. The allele was present in 13 of 37 corn crib populations, 8 of 16, and 6 of 21, in 1974, 1975, and 1976, respectively.

Analysis of the presence of the $Pgm\text{-}1^{e}$ allele in each of the regions over three years showed that most often the allele was present in an isolated corn crib population of a particular region, and nowhere else in that region. For instance, Paincourt-1976 consisted of samples from 5 different corn cribs, each with more than 35 animals, but only in the G. Belanger corn crib population was $Pgm\text{-}1^{e}$ present. Similar outcomes were found for Paincourt-1974-1975, East Chatham-1974, Wheatley-1974, Stoney Point-1975, and Central Essex-1976 populations.

The presence of $Pgm\text{-}1^{e}$ in Harrow-1974 was interesting in that it was found only in three nearby corn crib populations,

Bondy, D. Martin, and Pidgeon, and had not apparently spread to any of the other three corn crib populations of the area or to the barn population at Bondy.

The rare allele seemed generally widespread in the regions of Central Essex-1974 and Harrow-1975-1976; and was totally absent in Prairie Siding-1976 and Stoney Point-1974-1976.

Analysis of the three year data revealed that the Pgm-1^e allele persisted at Bondy barn and G. Belanger crib populations for two years and D. Martin and Maitre North populations for all three years. More often, however, the alleles fluctuated between being present and appearing absent. For example, for the Pidgeon population, the allele was present in 1974, absent in 1975, and present again in 1976. Several other combinations of the "present-absent" pattern were found at Bondy barn, Baillargeon, Belanger, and G. Belanger populations over three years and at Bondy, K. P. Martin, Parkes East, and Price North and South populations over two years. The Pgm-1^e allele was not detected in several corn crib populations: Rocheleau East, E. Comartin, Nussey, C. and N. R. Wilkinson over two years; and, Houle over three.

C The Pgm-2^b Allele

The frequency of Pgm-2^b ranged from zero to 0.2727 for the corn crib populations over the three years, with the highest frequency in the pooled data for 1975, as for Pgm-1^e. The allele was present in 6 of 37 corn crib populations, 7 of 16, and 8 of 21, in 1974, 1975, and 1976, respectively.

As with the analysis of Pgm-1^e in each of the regions over three years, Pgm-2^b proved to be most frequently in one isolated corn crib population and in no other population of that region. Stoney Point populations present an excellent example of this: Pgm-2^b was present only in the Houle population for all three years. Similar results occurred in Central Essex-1974-1976 and Paincourt-1975-1976 populations.

The Pgm-2^b allele seemed generally widespread in Harrow-1975-1976 populations, however, there were corn crib populations in which Pgm-2^b was not detected interspersed among populations with this rare allele.

The Pgm-2^b allele was found in populations inhabiting adjacent corn crib populations of Harrow-1974 and Wheatley-1974. The allele was not present in Paincourt-1974, East Chatham-1974, and Prairie Siding-1976 samples.

In the three year period of the study, Pgm-2^b usually did not appear to be present in the corn crib populations under consideration. Of the 8 populations without Pgm-2^b detected, 5 were monitored for two years, K. P. Martin, Rocheleau East, E. Comartin, Faubert, and N. R. Wilkinson; and, three were monitored for three years, Baillargeon, Belanger, and G. Belanger.

In a few cases the allele was present one year and absent the next or vice versa, as for example, in the crib and barn populations at Bondy, the crib populations at Parkes East, Price, D. Martin, Pidgeon, and Maitre North.

The allele persisted only at the C. Wilkinson population

over two years and Houle over three.

D The Pgm-2^C Allele

The frequency of Pgm-2^C ranged from zero to 0.3148 in individual populations over the three year period. In the pooled data, the highest frequency of Pgm-2^C occurred in the 1975 animals. This rare allele was not picked up in any of the corn crib populations of 1974; it was present in 4 of 16 corn crib populations and 3 of 21, in 1975 and 1976, respectively.

All three regions monitored in 1975 that were made up of three corn cribs or more showed Pgm-2^C isolated at only one corn crib population and nowhere else in that region. Of these three region samples, Stoney Point and Paincourt, along with one other, Prairie Siding, showed total absence of the allele in the following year, 1976. Only Harrow-1976 showed Pgm-2^C at an isolated population, Bondy barn.

Examination of the distribution of Pgm-2^C over the three year period revealed the allele to be apparently absent from most of the populations; when present in one year in a population, it was not found the following year (Bondy barn, Pidgeon, Houle, and G. Belanger).

E The Rare Alleles in Barn Populations

While most of the populations sampled were from corn cribs, two were barn populations in Harrow, Bondy, and D. Martin. A major difference between the two habitats is

that the barn offers a less disruptive existence for the mice. There are more places to hide in the barn and therefore the mice may escape capture when the corn is being moved and subsequently be protected from predators. As a result the barn mouse population may be more stable, with animals or their progeny remaining from year to year. Therefore, if a rare allele were to be introduced into a barn population, its probability of persisting should be greater than in a corn crib population, where there are drastic numerical bottlenecks.

A rare allele may appear in a population via two modes: mutation and migration. It is quite possible that the first PGM variants that occurred in the area did so by mutation. However, considering the duration of this study only, the variants probably migrated into the barn populations since they existed elsewhere in the region the year previous to appearing in the barn population.

An examination of the Bondy barn population, for instance, showed that Pgm-1^e was not present in 1974, but was present in the crib population of the same farm; in 1975, 7 of 30 animals were heterozygous for Pgm-1^e and Pgm-1^a in that barn population, however, unfortunately no sample was obtained from the crib; in 1976, Pgm-1^e was found again in the heterozygous form in the barn population, but not in the corn crib population (Table 19). It is possible that mice possessing the Pgm-1^e allele migrated from the crib to the barn sometime between 1974 and 1976; Pgm-1^e became established in the

barn population, but disappeared from the crib population.

The same type of situation with the Pgm-2^b allele occurred at Bondy (Table 20).

The highest allelic frequency of the region for Pgm-1^e and Pgm-2^b was observed in the D. Martin barn population, which unfortunately was monitored only in 1975.

Where many corn crib populations showed the rare alleles, if not disappearing from 1975 to 1976, then decreasing in frequency, the Bondy barn population showed Pgm-1^e and Pgm-2^b increasing in frequency. This increase is slight and although it may be the result of greater long term stability in the barn populations, it may also be simply a random fluctuation. More data from barn populations is required.

F Summary of the Three Rare Alleles

The three rare PGM alleles under consideration whenever observed were found widespread throughout the area with the alleles apparently appearing and disappearing from various populations over time.

A Hardy-Weinberg analysis revealed a significant deficiency of heterozygotes at both loci, Pgm-1 in the 1975 and 1976 samples and Pgm-2 in the 1974 and 1976 samples.

G Variance Analysis of PGM Data

The values of the genetic and Wahlund's variance were generally low because they depend on the allelic frequencies. In this case, Pgm-1^a and Pgm-2^a were the common alleles.

Table 21 summarizes the variance analysis of the mouse populations with respect to the erythrocytic Pgm-1 and Pgm-2 loci. The observed variance ranged from zero to 0.0145 and from zero to 0.0144 in Pgm-1 and Pgm-2, respectively. The sampling variance, if based on sampling from a large population, could account for all of the observed variance, leaving the value of the genetic variance at zero for 11 of 14 regional populations for Pgm-1 and 8 of 14 for Pgm-2.

The F test (Sokal and Rohlf, 1969) was performed on the erythrocytic PGM data to determine whether the observed variance was significantly different from the sampling variance. The results are summarized in Table 22. Of the F values determined for the regions, for the two loci over the three years, 5 of 28 were significant at the one percent level. In other words, for these 5 cases, the observed variance was significantly different from the sampling variance, which would be expected if the variance observed was not due only to sampling variance, but also Wahlund's variance.

On the basis of this evidence, therefore, population subdivision is possible. However, these results do not strongly support this hypothesis, and furthermore, of the F values determined for the kidney enzyme data (Table 11), only two were found significant. These results however are tempered with small sample size.

Consequently, in most cases, since sampling and observed variances are not significantly different, the population may very well be panmictic. However, on the other hand, the

Table 21: A summary of variances at Pgm-1 and Pgm-2 controlling the erythrocytic enzymes in 1974, 1975, and 1976 regional and pooled samples. (σo - observed variance; σs - sampling variance; f - Wahlund's variance).

		1974		1975		1976	
REGION		Pgm-1	Pgm-2	Pgm-1	Pgm-2	Pgm-1	Pgm-2
Harrow	No.	195	195	188	188	298	298
	σo	0.0001	0.0003	0.0044	0.0011	0.0046	0.0032
	σs	0.0003	0.0003	0.0020	0.0010	0.0010	0.0012
	f	0.0095	0.0368	0.0713	0.0255	0.0748	0.1004
Central Essex	No.	213	213	184	184	175	175
	σo	0.0005	0.00004	0.0145	0.00006	0.0001	0.0144
	σs	0.0013	0.0001	0.0011	0.00008	0.0001	0.0020
	f	0.0166	0.0200	0.2705	0.0078	0.0056	0.2093
Stoney Point	No.	206	206	86	86	364	364
	σo	0	0.0007	0.00008	0.0002	0	0.00005
	σs	0	0.0002	0.0002	0.0003	0	0.00004
	f		0.0233	0.0143	0.0134		0.0068
Paincourt	No.	48	48	123	123	223	223
	σo	0.00009	0	0.0007	0.0001	0.000004	0.00002
	σs	0.0004	0	0.0013	0.0003	0.00001	0.00005
	f	0.0096		0.1764	0.0107	0.0020	0.0108
East Chatham	No.	127	127	0	0	0	0
	σo	0.0002	0				
	σs	0.0005	0				
	f	0.0683					
Wheatley	No.	162	162	13	13	28	28
	σo	0.00001	0.0002				
	σs	0.00005	0.0004				
	f	0.0059	0.0156				
Total (pooled)	No.	960	960	594	594	1157	1157
	σo	0.0004	0.0004	0.0030	0.0008	0.0022	0.0036
	σs	0.0005	0.0002	0.0008	0.0005	0.0003	0.0006
	f	0.0337	0.0345	0.0795	0.0337	0.1106	0.1554

NOTE: In calculating variances for Pgm-2, Pgm-2[b] and Pgm-2[c] alleles were treated as one.

Table 22: F values for σ_o/σ_s for Pgm-1 and Pgm-2 controlling the erythrocytic enzyme in 1974, 1975, and 1976 regional and pooled samples. The asterisks (**) refer to a one percent level of significance of the F value.

	1974		1975		1976	
REGION	Pgm-1	Pgm-2	Pgm-1	Pgm-2	Pgm-1	Pgm-2
Harrow	0.3333	1.0000	0.0020	1.1000	4.6004**	2.6666**
Central Essex	0.3846	0.4000	13.1818**	0.7500	1	7.2000**
Stoney Point	0	3.5000**	0.4000	0.6666	0	1.2500
Paincourt	0.2250	0	0.5384	0.3333	0.4000	0.4000
East Chatham	0.4000	0				
Wheatley	0.2000	0.5000				
Pooled	0.8000	2.0000	3.7500**	1.6000	7.3333**	6.0000**

population may be subdivided with migration and/or selection decreasing observed variance.

Wahlund's variance is based on observed variance since, as discussed earlier, the sampling variance estimate is much higher than it should be because the samples are taken from small populations and in many cases, almost the entire population is typed. Therefore, genetic variance approaches observed variance.

The range of Wahlund's variance extended from zero to 0.2705 and from zero to 0.2093 for Pgm-1 and Pgm-2, respectively. For both loci the highest genetic and Wahlund's variances were for the Central Essex populations, as was the mean Wahlund's variance: 0.0975 and 0.0790 for Pgm-1 and Pgm-2, respectively. For the pooled data, Wahlund's variance was greatest for Pgm-1 in 1975 and for Pgm-2 in 1976.

In summary, the overall population is probably not panmictic, but subdivided. This is suggested by the Hardy-Weinberg equilibrium deviations, population sampling and variance.

H Migration Rate and Breeding Unit Size

Migration rates required to maintain the Pgm-1 and Pgm-2 allelic frequencies are summarized in Table 23. These estimates are based on founder population sizes of 10 and 20 and on Wahlund's variance calculated from the observed variance. These calculations were done as they were for the kidney enzyme data previously discussed. The range of the

Table 23: Estimated migration rates to maintain Pgm-1 and Pgm-2 allelic frequencies in the regional and pooled populations of 1974, 1975, and 1976. (N - effective population size).

		1974		1975		1976	
REGION	N	Pgm-1	Pgm-2	Pgm-1	Pgm-2	Pgm-1	Pgm-2
Harrow	10	2.6065	0.6543	0.3256	0.9553	0.3092	0.2240
	20	1.3032	0.3271	0.1628	0.4776	0.1546	0.1120
Central Essex	10	1.4810	1.2250	0.0674	3.1801	4.4393	0.0946
	20	0.7405	0.6125	0.0337	1.5900	2.2193	0.0472
Stoney Point	10		1.0479	1.7232	1.8406		3.6514
	20		0.5239	0.8616	0.9203		1.8257
Paincourt	10	2.5260		0.1167	2.3114	12.4750	2.2898
	20	1.2630		0.0583	1.1557	6.2375	1.1449
East Chatham	10	0.3410					
	20	0.1705					
Wheatley	10	4.2122	1.5775				
	20	2.1061	0.7887				
Pooled	10	0.7168	0.6996	0.2894	0.7168	0.2010	0.1358
	20	0.3584	0.3498	0.1447	0.3584	0.1005	0.0679
3 year mean (pooled)	10	0.4024	0.5174				
	20	0.2012	0.2587				

estimated migration rate for the different regional populations based on Pgm-1 data for N=10 was 0.0674 to 12.4750 and for N=20, 0.0337 to 6.2375; based on Pgm-2, for N=10 it was 0.0946 to 3.6514 and for N=20, 0.0472 to 1.8257. Many of these estimates of migration rate were much above a reasonable rate for both population sizes: 7 of 12 cases for Pgm-1 and 10 of 12 for Pgm-2.

While the migration rate estimates to maintain the regional allelic frequencies were generally higher than what realistically the migration rates could be, that for the pooled data was reasonable for both loci for all three years at N=20 and for 1975 and 1976 at N=10, for Pgm-1; and, for 1976 at N=10, for Pgm-2. The mean migration rate values for Pgm-1 were 0.4024 (N=10) and 0.2012 (N=20) and for Pgm-2, 0.5174 (N=10) and 0.2587 (N=20). The migration rate values for the pooled population based on the blood PGM were not unlike those based on the kidney enzymes (Table 16).

It is possible, then, that migration alone is maintaining allelic frequencies in the population. There seems no need to invoke the theory of selection to explain the results.

Another consideration is to calculate the values of effective population size, N, to determine if N is indeed within the range used. The formula employed was based on Wahlund's variance,

$$f = \frac{1}{2N}$$

where f is Wahlund's variance and N is effective population size (Table 24). The mean effective population size estimate for three year data for Pgm-1 was 8.6 and for Pgm-2 was 10.9. This suggests that the effective population size is indeed close to the lower limit, 10, used.

In summary, migration alone may be maintaining allelic frequencies in the population and the breeding unit size based on f appears to be about 10.

I Residual Population

From the data available (Tables 19 and 20), it is evident that the rare PGM alleles fluctuate in appearance throughout the area and it is possible that migration played a large role in this occurrence. For example, Pgm-1^e was found at the D. Martin and Maitre North populations for three years and Pgm-2^b at the Houle population without genetic drift eliminating the rare alleles. On the other hand, where three year data were also available, the rare alleles were present and absent, for example, Pgm-1^e was present in the Pidgeon-1974 population, absent in 1975, possibly due to genetic drift, but present again in 1976, at a higher frequency. The only population in the Harrow region in 1975 with the Pgm-1^e allele was K. P. Martin and upon calculation of the migration rate necessary from the K. P. Martin population to the Pidgeon population (m=1.7281) to re-establish the rare allele at the Pidgeon population in 1976, it was found to be unrealistically high. Similar results were found for most other cases of the

Table 24: Effective population size estimates from the equation F=1/2N for the pooled populations of each of three years and three years combined.

YEAR	Pgm-1	Pgm-2
1974	14.8	14.5
1975	6.3	14.8
1976	4.5	3.2
Mean for 3 year data	8.6	10.9

rare alleles fluctuating in appearance.

One possible explanation is then that a residual population exists formed from mice which have escaped capture and survived the summer. These mice, or more probably, their descendants move back into the corn cribs after they have been emptied and not necessarily into those from which they came. This would explain the sudden appearance of a rare allele in a corn crib population one year, when it was not there the year before, or in any nearby population, as with the Baillargeon population with respect to Pgm-1^e.

J Additional Zones of PGM Activity

Many regions of PGM activity have been identified in the human (Spencer et al., 1964; Parrington et al., 1968), while in the mouse only two zones, made up of several components, have been described (Shows et al., 1969; Miner and Wolfe, 1972). During the course of this study, two regions of PGM activity, other than PGM-1 and PGM-2, were identified in mouse erythrocytes. For the purposes of this survey, these were designated "PGM-3" and "PGM-4". Both of these regions of electrophoretic activity fall between PGM-1 and PGM-2. These two new PGM regions have been discussed previously (see Methods) and are illustrated in Figure 8. From Table 25, it is evident that the "PGM-3B" band was most frequently found and from Table 26, that "PGM-4" was more often absent than present.

Table 25: A summary of phenotypes of "PGM-3" in 1974 *Mus musculus* populations.

REGION	SAMPLE SIZE	A	AB	B	O
Harrow	46	9	9	24	4
Sandwich West	3	1	2	0	0
Central Essex	65	11	18	25	11
Stoney Point	34	5	11	17	1
Paincourt	5	0	1	0	4
East Chatham	64	17	16	19	12
Wheatley	63	8	17	32	6
Pooled for all regions	280	51	74	117	38

Table 26: A summary of phenotypes of "PGM-4" in 1974 *Mus musculus* populations.

REGION	SAMPLE SIZE	PRESENT	ABSENT
Harrow	46	12	34
Sandwich West	3	1	2
Central Essex	65	7	27
Paincourt	5	0	5
East Chatham	64	20	44
Wheatley	63	22	41
Pooled for all regions	280	76	204

K Discussion

The final outcome of genetic drift, particularly on small populations, is always fixation of one allele and loss of others, unless there are intervening pressures, which are: mutation, migration, and selection (Cavalli-Sforza and Bodmer, 1971). Since the breeding unit sizes of the populations being studied are small, it would not be surprising to find the elimination of the rare PGM alleles. This, however, has not happened. Furthermore, if populations are small, with rare alleles present, the alleles are expected to be concentrated in several of the populations only, with their frequency high unless selection is occurring (Cavalli-Sforza and Bodmer, 1971). In the populations studied, the rare alleles were found widespread throughout the study area, but at low frequency for up to three years.

Gene flow could account for the rare alleles present at low frequencies and also appearing for the first time in new populations, thereby spreading through the area. Of course, mutation could also explain the new appearance of a rare allele, and probably does account for the appearance of the first PGM rare alleles in the population, but it is unlikely that mutation is introducing, maintaining, or restoring variation during the time span of this study. As established, the theory of selection need not be invoked as explanation for the allelic frequencies observed, since migration alone is sufficient to explain the results obtained.

In summary, the PGM data suggest that the overall mouse

population studied is not panmictic, but subdivided into demes with genetically effective sizes of 10 animals, with a residual population contributing to each of the demes after they have been disrupted. Migration between the demes and from the residual population to the demes quite likely plays a large role in maintaining the rare alleles in the population.

V DISCUSSION AND CONCLUSIONS

The purpose of this present study was to determine the relative roles that mutation, migration, and selection play in counteracting random genetic drift and thereby resulting in the allelic frequencies of 7 polymorphic loci observed.

Much research has been done on Mus Musculus, however, still major breaks in the information on the genetic aspects of natural populations of the house mouse exist. Many investigators have dealt with the question of genetic variation within inbred mice under laboratory conditions (Chapman et al., 1970; DeLorenzo and Ruddle, 1970; Nichols et al., 1973); fewer investigations have dealt with genetic variation within wild mouse populations (Petras, 1967a, b; Petras et al., 1969; Selander et al., 1969; Selander, 1970a, b; Roderick et al., 1971).

In attempting to solve the aforementioned problem, 12 loci were examined: all of these were examined for kidney enzymes and two for erythrocytic enzymes. Five loci were found monomorphic. Of the two loci examined controlling erythrocytic enzymes, two new variants, Pgm-1^e and Pgm-2^c, were discovered. Breeding studies indicated that these new variants were alleles at their respective loci (see Appendix I).

With the information available from the biochemical markers, specifically, phenotypic frequencies, the problem

was approached in several ways.

First, genic variability was measured by different methods to determine whether the Mus populations considered were similar to others studied. One such measure is genic heterozygosity. The average heterozygosity for the Mus population under consideration, 0.0886, fell well within the range of other investigations (Roderick et al., 1971) and was also not unlike the estimates for other species: Drosophila (O'Brien and MacIntyre, 1969; Prakash, 1969; Prakash et al., 1969); the horseshoe crab (Selander et al., 1970); and, the human (Lewontin, 1967; Harris, 1969).

Another measure of genic variability is genetic distance. Wright (1943) reported that if populations are "isolated by distance", local differentiation of gene frequenices is expected. Generally, Nei and Imaizumi (1966a, b) found the more geographical separation there was between Japanese subpopulations, the more different they were in terms of gene frequency. Similar results were obtained for Drosophila, fish, reptile, and rodent (Peromyscus) populations (Chakraborty et al., 1977). In the present study, only a slight correlation, at the 5 percent level of significance, was found between geographic and genetic distances for the Mus population.

It seemed, therefore, that pressures were counteracting random genetic drift, at least to some degree. Variance analysis was then done to determine whether this was the case.

Considerable attention has been paid to allelic frequency variance for Mus populations (Selander, 1970a, b). Using

Wahlund's variance, a measure of allelic frequency differences between populations, attempts to determine the extent of population subdivision or inbreeding have been made (Petras, 1967a; Rasmussen, 1964) with varying results. Petras (1967a) and Petras et al. (1969) did observed heterozygote deficiencies which they attributed to inbreeding or population subdivision. On the other hand, Roderick et al. (1971) did not detect any evidence for inbreeding from numerical deficiencies of heterozygotes, however, the sample size of this study was small. Selander (1970b) observed heterogeneity in allelic frequencies among samples from different farms in the same region and from different barns on the same farm. Population subdivision and genetic drift, he felt, would explain these results. Selander (1970a) also felt that natural selection did have a role in maintaining certain allelic frequencies, however, direct or even circumstantial evidence to substantiate this claim is difficult to obtain.

In the present study, numerical deficiencies of heterozygotes existed at all loci in various samples and considerable heterogeneity in allelic frequencies was evident. These, together with ecological data, suggest that the overall population was subdivided, and not panmictic. This is consistent with Petras's (1967a) suggestion that the breeding unit size was small for Mus populations, perhaps as low as 10 animals. This conclusion was based on results derived from the combination of the coefficient of inbreeding estimated from empirical studies of two polymorphic loci, Es-2 and Hb,

studied in mice collected from southeastern Michigan, with such ecological data as estimates of migration.

In the past, migration between Mus populations has been reported as infrequent (Brown, 1953; Rowe et al., 1963; Southern and Laurie, 1946). From a stochastic model developed by Topping (1975) to account for the polymorphism at the T locus, a migration rate of 5 percent explained the empirical data well. In the present study, a mean migration rate of 0.2981 based on an effective population size of 10 could account for the allelic frequencies observed for 7 polymorphic loci controlling renal enzymes. One possible explanation for the difference between the values of migration rate previously reported and those of this study is that the latter were not based on direct observation, but calculated from two parameters, the Wahlund's variance, possibly an overestimate, and the effective population size, an estimate also not necessarily exact.

The conclusion was therefore, that neither mutation nor selection needed to be invoked to explain the data. Migration alone, in most cases, is sufficient to counteract random genetic drift.

The present study also leads to the following general model of Mus populations. The mice do not form a panmictic population, but one that appears subdivided because of their habitat preference, namely corn cribs or barns. These habitats are scattered with no distinct pattern throughout the area. The distances between them vary considerably. There

appears to be some gene flow between these corn crib populations, but again, a pattern is not evident. Furthermore, the ages of the mice in each corn crib population vary, so there may be several generations of mice per population. These characteristics must be taken into consideration when choosing a suitable population model from the many described in the literature.

A model for population structure based on partially isolated subpopulations was proposed by Wright (1943) and was called the "island model". According to this model, every subpopulation exchanges genes equally with every other. The principal drawback of applying the "island model" to the population studied is that it does not take geographic distance into account, that is, the fact that more distant subpopulations are less likely to exchange individuals with each other than closer subpopulations.

Several other models have been proposed, all of which consider geographic distance. There are two types: a continuous model, in which population density is constant at any point (Wright, 1943; 1946; 1951; Malécot, 1967) and a discontinuous model, in which the population is grouped at the nodes of a lattice (Kimura and Weiss, 1964). Both of these models exist in at least two forms: a linear one with a one-dimensional distribution of a population and a two-dimensional one with no barriers against diffusion of the population in any direction. The discontinuous model is often referred to as the "stepping-stone" model and in the two-dimensional form,

it appears to best fit the southwestern Ontario mouse population of this study. One expectation of this model is that geographic variation in gene frequency should increase with distance. Looking back to Figure 11, comparisons of Wahlund's variance for three regions separated by various geographic distances did not prove this to be true for any loci considered nor was the migration rate smaller for regions separated by greater distance as opposed to smaller.

While the two-dimensional "stepping-stone" model appears to best fit that of the population structure under consideration, there are several objections to it. These include the fact that the model does not take into account that the mice are irregularly distributed over the area in clusters of different size and separated by varying distance. Also, migration rates between subpopulations are quite variable; and, neither natural selection nor age structure are taken into account.

In summary, most studies of natural populations of Mus musculus have resulted in a population structure theory of tribal or territorial subdivision, with close interbreeding within the tribes, little inter-tribal migration, and small genetically effective tribal unit sizes (Lewontin and Dunn, 1960; Crowcroft and Rowe, 1963; Anderson and Hill, 1965; Reimer and Petras, 1968; Rowe and Redfern, 1969; Selander, 1970a). With the integration of the information derived from the various concepts and measures considered in this present study, the model of population structure for Mus musculus

populations of southwestern Ontario agrees with the aforementioned.

VI SUMMARY

1. Mice (*Mus musculus*) were collected from 52 corn cribs and two barns located on farms throughout southwestern Ontario.

2. Twelve biochemical markers were examined electrophoretically in kidney homogenates of the mouse and two in hemolysates: 7 loci were found polymorphic (*Id-1*, *Mod-1*, *Got-2*, *Dip-1*, *Gpd-1*, *Pgm-1*, and *Pgm-2*); 5 loci were found monomorphic (*Got-1*, *Ipo-1*, *Trip-1*, *Mpi-1*, and *Ldh-1*).

3. Two previously unreported alleles, $Pgm\text{-}1^{e}$ and $Pgm\text{-}2^{c}$, were found in the samples, both at very low frequencies, generally lower than 0.04. Based on breeding studies, these two new electrophoretic patterns were not the result of non-genetic factors, but, very likely alleles at their respective loci (see Appendix I).

4. Departures from Hardy-Weinberg equilibrium as a result of numerical deficiencies of heterozygotes occurred at 5 of 7 loci studied.

5. Mean genic heterozygosity was established at 0.0886, a value well within the range of other investigations of *Mus* and other species.

6. Genetic distance values suggested that there was as much genetic differentiation between populations of a single region, as between populations of different regions. There appeared to be a weak correlation (at the 5 percent level of significance) between geographic and genetic distances.

7. Variance analysis was performed and for the pooled samples, in most cases, sampling variance was smaller than the observed, however, generally not significantly smaller, suggesting the population under consideration to be panmictic. However, observed variance could be decreased by migration and/or selection, with the population actually subdivided.

8. All regions with an appreciable sample size displayed heterogeneity of Wahlund's variance at all loci studied except Pgm-1 and Pgm-2. Based on kidney homogenate data, the mean value of Wahlund's variance was 0.0624.

9. Based on kidney homogenate data and an effective population size of 10, the mean migration rate estimate to maintain the appropriate values of Wahlund's variance was 0.2981; based on hemolysate data, these estimates were 0.4024 (Pgm-1) and 0.5174 (Pgm-2). It is possible that migration alone is maintaining uniformity in allelic frequencies. There seems, in most cases, no need to invoke the theories of natural selection nor mutation.

11. Effective population size estimates, migration rate estimates, variance analysis, departures from Hardy-Weinberg equilibrium, and direct observation of natural populations support a model involving population subdivision with fairly high turnover involving a residual population.

APPENDIX I

PHOSPHOGLUCOMUTASE (PGM) BREEDING STUDIES

Shows et al. (1969) described two allelic forms, Pgm-1^a and Pgm-1^b, in Mus musculus erythrocytes, segregating at a single autosomal locus. Segregation of alleles at Pgm-2, also an autosomal locus was originally observed only in feral mice (Shows et al., 1969). Subsequently, a variant allele at the Pgm-2 locus was discovered in the inbred strain of SM/J (Chapman et al., 1970. The two PGM loci are not linked: Pgm-1 is on linkage group (LG) XVII, chromosome 5 (Hutton and Roderick, 1970) and Pgm-2 is on LG VIII, chromosome 4 (Chapman et al., 1970).

Concurrent with the above study, Miner and Wolfe (1972) described two new alleles, Pgm-1^c and Pgm-1^d at the Pgm-1 locus in mice. They used two modified buffer systems outlined by Spencer et al. (1964) and Shows et al. (1969), and found that the alleles, Pgm-1^a and Pgm-1^b, varied in their appearance electrophoretically, depending on the buffer used. Using Spencer's buffer system, they found two new alleles, which they described as Pgm-1^c and Pgm-1^d, and claimed these alleles to be previously indistinguishable from Pgm-1^b and Pgm-1^a, respectively. Apparently, the Show's buffer system could not separate the four banding patterns because of

smearing or streaking. The Spencer's buffer system did not totally eliminate the problem, but did offer sharper banding patterns.

The two new alleles described by Miner and Wolfe (1972) could not be distinguished in the present study, even with their modified buffer systems. However, using Spencer's buffer system (see Methods), two previously undescribed alleles were found in wild mice: the bands controlled by Pgm-1^e migrated anodally to the bands controlled by Pgm-1^a and the bands controlled by Pgm-2^c, migrated cathodally to those controlled by Pgm-2^b.

Breeding studies were conducted to determine whether the PGM patterns were in fact the result of new alleles at the Pgm-1 and Pgm-2 loci. Other explanations such as non-genetic modifications of the PGM electrophoretic patterns or mutations at another locus could also account for the observations.

Chi-square analysis, based on the hypothesis of a single-locus, two allele mode of inheritance was performed on the breeding study data and the results are summarized in Tables 27, 28, and 29.

For examination of Pgm-1^e, 6 different types of matings were set up and these are presented in Table 27. The offspring phenotypes from all matings, but one, agreed with Mendelian expectations. Mating type 3 (PGM-1AE X PGM-1AE) revealed a significant deficiency of PGM-1A homozygotes at the 5 percent level. Table 28 presents the data for each of the 6 matings that contributed to that mating type. Two of

Table 27: Summary of Pgm-1 "mating - offspring" results. X^2 analysis is based on a one locus multi-allele hypothesis.

MATING TYPE ♂ X ♀	NO. OF SIBSHIPS	NO. OF INDIVIDUALS	OFFSPRING PHENOTYPES aa	ab	bb	be	ee	ae	X^2	d.f.	P
1. ee X aa	1	30						30			
2. aa X ae	5	39	20					19	0	1	
ae X aa	6	63	25					38	2.2857	1	
3. ae X ae	6	117	17				37	63	7.5298	2	0.05
4. aa X bb	1	22		22							
5. ae X bb	2	27		19		8			3.7037	1	
bb X ae	1	17		10		7			0.2352	1	
6. aa X aa	2	18	18								

Table 28: Summary of (ae X ae) Pgm-1 "mating - offspring" results. X^2 analysis is based on a one locus multi-allele hypothesis.

MATING NUMBER	NO. OF INDIVIDUALS	OFFSPRING PHENOTYPES aa	ae	ee	X^2	d.f.	P
1.a	16	1	12	3	4.5000	2	
b	5	1	2	2	0.6000	2	
2.	8	1	3	4	2.7500	2	
3.	32	5	20	7	2.2500	2	
4.a	15	0	8	7	6.2500	2	0.05
b	12	4	5	3	0.4999	2	
5.	7	1	2	4	3.8570	2	
6.	22	4	11	7	0.8181	2	
TOTAL	117	17	63	37	7.5298	2	0.05

Table 29: Summary of Pgm-2 "mating - offspring" results. X^2 analysis is based on a one a one locus multi-allele hypothesis.

MATING TYPE ♂ X ♀	NO. OF SIBSHIPS	NO. OF INDIVIDUALS	OFFSPRING PHENOTYPES aa	ab	bb	bc	bb	ac	X^2	d.f.	P
1. ab X ab	1	8	1	5	2				0.7500	2	
2. aa X ab	1	22	11	11					0.0454	1	
3. ac X aa	1	15	10					5	1.0666	1	
4. bb X ac	2	18		12		6			1.3888	1	

the 6 matings (mating 1a and 4a) showed a deficiency of PGM-1A homozygotes, however, only one at the 5 percent level of significance. In both of these cases, the original parents were typed again and mated again to eliminate the possibility of error. Typing of the parents proved to be correct and the offspring from the second mating did not show a deficiency of PGM-1A homozygotes.

Table 29 summarizes the mating studies for Pgm-2. Four different mating types were set up, with none of the mating results deviating from Mendelian expectations based on the hypothesis of a new allele at the Pgm-2 locus.

Further breeding studies involving mice with phenotypes PGM-1AE X PGM-1BB showed that none of the 44 offspring had electrophoretic patterns with three bands (i.e. PGM-1A, PGM-1B, and PGM-1E), a result that would be expected if $Pgm\text{-}1^{e}$ were not an allele at the Pgm-1 locus. The probability of not finding a three banded pattern, in this case, is $(1/2)^{44}$ or 5.68×10^{-14}.

Similar results were obtained for Pgm-2 from mating type PGM-2BB X PGM-2AC. None of the 18 offspring showed a three banded electrophoretic pattern (i.e. PGM-2A, PGM-2B, and PGM-2C) and therefore, $Pgm\text{-}2^{c}$ can be considered another allele at its Pgm-2 locus. The probability of not detecting a three banded pattern, in this case, is $(1/2)^{18}$ or 3.00×10^{-6}.

Since the two rare patterns did occur consistently in the offspring of the mating studies, they were not the result of non-genetic factors, but, of alleles at their respective loci.

APPENDIX II

TABLE OF PHENOTYPES

Table 30: A summary of phenotypes for each of the polymorphic loci controlling renal enzymes for all corn crib and regional populations monitored during the years of 1973 and 1974.

1973

SITE/REGION	Id-1		Mod-1		Got-2		Dip-1	
Arner	5	a	2	a	4	a	0	a
	0	b	0	b	0	b	4	b
	0	ab	3	ab	1	ab	1	ab
Bondy	3	a	2	a	4	a	1	a
	2	b	2	b	1	b	4	b
	0	ab	2	ab	0	ab	0	ab
Hutchins	1	a	0	a	0	a	0	a
	0	b	1	b	0	b	1	b
	0	ab	0	ab	1	ab	0	ab
D. Martin	0	a	3	a	4	a	2	a
	2	b	0	b	0	b	1	b
	2	ab	2	ab	0	ab	1	ab
Parkes	0	a	3	a	1	a	0	a
	1	b	0	b	0	b	0	b
	0	ab	0	ab	0	ab	1	ab
HARROW	9	a	10	a	13	a	3	a
	5	b	3	b	1	b	10	b
	2	ab	7	ab	2	ab	3	ab

Table 30: continued

SITE/REGION	Id-1		Mod-1		Got-2		Dip-1	
McKim	2	a	5	a	2	a	2	a
	1	b	0	b	2	b	3	b
	2	ab	1	ab	1	ab	0	ab
Maitre North	1	a	3	a	3	a	1	a
	3	b	4	b	3	b	2	b
	4	ab	1	ab	1	ab	4	ab
Maitre South	6	a	14	a	12	a	3	a
	7	b	5	b	7	b	13	b
	10	ab	4	ab	2	ab	5	ab
Price North	7	a	9	a	9	a	5	a
	3	b	3	b	2	b	3	b
	3	ab	1	ab	1	ab	4	ab
CENTRAL ESSEX	16	a	31	a	26	a	11	a
	14	b	12	b	14	b	21	b
	19	ab	7	ab	5	ab	13	ab
Damphouse	3	a	7	a	1	a	5	a
	2	b	1	b	4	b	0	b
	1	ab	0	ab	0	ab	0	ab
E. Comartin	5	a	12	a	4	a	2	a
	4	b	3	b	5	b	3	b
	5	ab	1	ab	3	ab	7	ab

Table 30: continued

SITE/REGION	Id-1		Mod-1		Got-2		Dip-1	
Houle	62	a	87	a	68	a	41	a
	17	b	26	b	45	b	48	b
	49	ab	20	ab	11	ab	35	ab
Baillargeon	2	a	7	a	2	a	1	a
	1	b	1	b	2	b	1	b
	3	ab	0	ab	0	ab	2	ab
Nussey	2	a	3	a	0	a	0	a
	0	b	1	b	1	b	1	b
	1	ab	0	ab	1	ab	1	ab
STONEY POINT	74	a	116	a	75	a	49	a
	24	b	32	b	57	b	53	b
	59	ab	21	ab	15	ab	45	ab
Pinnsonneault	1	a	4	a	4	a	2	a
	3	b	2	b	3	b	5	b
	5	ab	4	ab	1	ab	1	ab
PRAIRIE SIDING	1	a	4	a	4	a	2	a
	3	b	2	b	3	b	5	b
	5	ab	4	ab	1	ab	1	ab
Gagnier	3	a	5	a	2	a	1	a
	2	b	0	b	2	b	3	b
	0	ab	0	ab	1	ab	1	ab

Table 30: continued

SITE/REGION	Id-1		Mod-1		Got-2		Dip-1	
G. Belanger	3	a	3	a	2	a	0	a
	0	b	0	b	0	b	3	b
	0	ab	0	ab	1	ab	0	ab
Roy III	4	a	9	a	6	a	4	a
	3	b	0	b	1	b	1	b
	1	ab	0	ab	0	ab	2	ab
PAINCOURT	10	a	17	a	10	a	5	a
	5	b	0	b	3	b	7	b
	1	ab	0	ab	2	ab	3	ab
Van K	2	a	4	a	3	a	1	a
	0	b	0	b	1	b	2	b
	2	ab	0	ab	0	ab	1	ab
Bodnar	1	a	1	a	1	a	0	a
	0	b	0	b	0	b	0	b
	0	ab	0	ab	0	ab	1	ab
Brown	5	a	8	a	3	a	2	a
	3	b	0	b	2	b	6	b
	2	ab	3	ab	3	ab	0	ab
EAST CHATHAM	8	a	13	a	7	a	3	a
	3	b	0	b	3	b	8	b
	4	ab	3	ab	3	ab	2	ab

Table 30: continued

SITE/REGION	Id-1	Mod-1	Got-2	Dip-1
POOLED SAMPLES	118 a	191 a	135 a	73 a
	54 b	49 b	81 b	104 b
	90 ab	42 ab	28 ab	67 ab

1974

SITE/REGION	Id-1	Mod-1	Got-2	Dip-1	Gpd-1	Pgm-1	Pgm-2
Bondy (crib)	27 a	30 a	28 a	17 a	11 a	39 a	40 a
	2 b	1 b	0 b	23 b	19 b	0 e	0 b
	5 ab	0 ab	12 ab	0 ab	10 ab	1 ae	0 ab
Bondy (barn)	4 a	4 a	2 a	1 a	4 a	5 a	5 a
	0 b	0 b	0 b	4 b	1 b	0 e	0 b
	0 ab	0 ab	3 ab	0 ab	0 ab	0 ae	0 ae
D. Martin (crib)	14 a	15 a	17 a	9 a	8 a	21 a	22 a
	3 b	1 b	1 b	13 b	11 b	0 e	0 b
	2 ab	1 ab	4 ab	0 ab	3 ab	1 ae	0 ab
Pidgeon	4 a	4 a	2 a	1 a	1 a	4 a	2 a
	0 b	0 b	0 b	3 b	1 b	0 e	0 b
	0 ab	0 ab	2 ab	0 ab	2 ab	0 ae	2 ab
Richardson	3 a	4 a	5 a	1 a	1 a	7 a	7 a
	0 b	1 b	1 b	6 b	3 b	0 e	0 b
	1 ab	0 ab	1 ab	0 ab	3 ab	0 ae	0 ab

Table 30: continued

SITE/REGION	Id-1		Mod-1		Got-2		Dip-1		Gpd-1		Pgm-1		Pgm-2	
Bennett	10	a	5	a	10	a	3	a	3	a	12	a	11	a
	1	b	1	b	1	b	8	b	5	b	0	e	0	b
	0	ab	0	ab	1	ab	1	ab	4	ab	0	ae	1	ab
Parkes	1	a	1	a	1	a	1	a	0	a	1	a	1	a
	0	b	0	b	0	b	0	b	1	b	0	e	0	b
	0	ab	0	ab	0	ab	0	ab	0	ab	0	ae	0	ab
HARROW	63	a	63	a	65	a	33	a	28	a	89	a	88	a
	6	b	4	b	3	b	57	b	41	b	0	e	0	b
	8	ab	1	ab	23	ab	1	ab	22	ab	2	ae	3	ab
Walters	4	a	5	a	2	a	1	a	1	a	3	a	5	a
	0	b	0	b	0	b	4	b	2	b	0	e	0	b
	1	ab	0	ab	3	ab	0	ab	2	ab	2	ae	0	ab
SANDWICH WEST	4	a	5	a	2	a	1	a	1	a	3	a	5	a
	0	b	0	b	0	b	4	b	2	b	0	e	0	b
	1	ab	0	ab	3	ab	0	ab	2	b	2	ae	0	ab
Chapo	4	a	2	a	2	a	0	a	2	a	3	a	4	a
	0	b	0	b	2	b	4	b	1	b	0	e	0	b
	0	ab	1	ab	0	ab	0	ab	1	ab	1	ae	0	ab
Rocheleau East	5	a	5	a	2	a	1	a	0	a	8	a	8	a
	0	b	0	b	4	b	6	b	5	b	0	e	0	b
	1	ab	0	ab	2	ab	1	ab	3	ab	0	ae	0	ab

Table 30: /continued

SITE/REGION	Id-1	Mod-1	Got-2	Dip-1	Gpd-1	Pgm-1	Pgm-2
Rocheleau West	2 a	2 a	4 a	0 a	3 a	6 a	6 a
	0 b	0 b	1 b	5 b	2 b	0 e	0 b
	3 ab	2 ab	1 ab	1 ab	1 ab	0 ae	0 ab
McKim	2 a	1 a	3 a	0 a	1 a	3 a	3 a
	0 b	0 b	0 b	3 b	0 b	0 e	0 b
	0 ab	0 ab	0 ab	0 ab	2 ab	0 ae	0 ab
Maitre North	43 a	37 a	15 a	6 a	7 a	49 a	50 a
	0 b	3 b	11 b	39 b	27 b	0 e	0 b
	6 ab	5 ab	24 ab	5 ab	13 ab	1 ae	0 ab
Maitre South	20 a	23 a	6 a	3 a	4 a	21 a	24 a
	0 b	0 b	4 b	21 b	10 b	0 e	0 b
	4 ab	0 ab	14 ab	0 ab	9 ab	3 ae	0 ab
Price North	11 a	10 a	2 a	0 a	3 a	10 a	12 a
	0 b	2 b	4 b	10 b	7 b	0 e	0 b
	1 ab	0 ab	6 ab	2 ab	2 ab	2 ae	0 ab
Price South	4 a	3 a	3 a	0 a	1 a	7 a	7 a
	2 b	1 b	1 b	7 b	4 b	0 e	0 b
	1 ab	0 ab	3 ab	0 ab	2 ab	0 ae	0 ab
CENTRAL ESSEX	91 a	83 a	37 a	10 a	21 a	107 a	114 a
	2 b	6 b	27 b	95 b	56 b	0 e	0 b
	16 ab	8 ab	50 ab	9 ab	33 ab	7 ae	0 ab

Table 30: continued

SITE/REGION	Id-1		Mod-1		Got-2		Dip-1		Gpd-1		Pgm-1		Pgm-2	
E. Comartin	6	a	6	a	3	a	2	a	2	a	8	a	8	a
	0	b	0	b	1	b	6	b	3	b	0	e	0	b
	0	ab	0	ab	4	ab	0	ab	3	ab	0	ae	0	ab
L. Comartin	6	a	7	a	3	a	2	a	2	a	8	a	8	a
	0	b	0	b	1	b	5	b	3	b	0	e	0	b
	0	ab	0	ab	4	ab	1	ab	2	ab	0	ae	0	ab
Houle	31	a	33	a	35	a	15	a	10	a	44	a	42	a
	0	b	5	b	3	b	28	b	24	b	0	e	0	b
	8	ab	2	ab	6	ab	1	ab	10	ab	0	ae	2	ab
Baillargeon	4	a	3	a	5	a	0	a	2	a	5	a	5	a
	0	b	0	b	0	b	5	b	2	b	0	e	0	b
	1	ab	2	ab	0	ab	0	ab	1	ab	0	ae	0	ab
Nussey	1	a	4	a	1	a	0	a	1	a	4	a	4	a
	0	b	0	b	1	b	4	b	2	b	0	e	0	b
	2	ab	0	ab	2	ab	0	ab	1	ab	0	ae	0	ab
STONEY POINT	48	a	53	a	47	a	19	a	17	a	69	a	67	a
	0	b	5	b	6	b	48	b	34	b	0	e	0	b
	11	ab	4	ab	16	ab	2	ab	17	ab	0	ae	2	ab
Belanger	2	a	3	a	1	a	2	a	3	a	7	a	7	a
	1	b	0	b	4	b	5	b	3	b	0	e	0	b
	2	ab	0	ab	2	ab	0	ab	1	ab	0	ae	0	ab
G. Belanger	0	a	1	a	1	a	0	a	0	a	2	a	2	a
	1	b	0	b	1	b	2	b	0	b	0	e	0	b
	1	ab	0	ab	0	ab	0	ab	2	ab	0	ae	0	ab

Table 30: continued

SITE/REGION	Id-1		Mod-1		Got-2		Dip-1		Gpd-1		Pgm-1		Pgm-2	
Faubert	4	a	5	a	3	a	3	a	2	a	6	a	6	a
	0	b	0	b	2	b	2	b	3	b	0	e	0	b
	1	ab	0	ab	1	ab	1	ab	1	ab	0	ae	0	ab
PAINCOURT	6	a	9	a	5	a	5	a	5	a	15	a	15	a
	2	b	0	b	7	b	9	b	6	b	0	e	0	b
	4	ab	0	ab	3	ab	1	ab	4	ab	0	ae	0	ab
Communication Road	6	a	6	a	2	a	5	a	2	a	9	a	9	a
	1	b	1	b	1	b	4	b	4	b	0	e	0	b
	2	ab	2	ab	6	ab	0	ab	3	ab	0	ae	0	ab
Snobdelen	8	a	8	a	2	a	4	a	4	a	12	a	12	a
	0	b	0	b	4	b	8	b	4	b	0	e	0	b
	4	ab	4	ab	6	ab	0	ab	4	ab	0	ae	0	ab
Nash Gravel Pit	4	a	4	a	0	a	0	a	0	a	5	a	5	a
	0	b	0	b	2	b	5	b	1	b	0	e	0	b
	1	ab	1	ab	3	ab	0	ab	3	ab	0	ae	0	ab
Nash	5	a	5	a	4	a	0	a	0	a	6	a	7	a
	0	b	0	b	2	b	6	b	1	b	o	e	0	b
	2	ab	2	ab	1	ab	1	ab	3	ab	1	ae	0	ab
Brown	6	a	6	a	3	a	0	a	1	a	13	a	13	a
	3	b	3	b	3	b	13	b	6	b	0	e	0	b
	3	ab	3	ab	7	ab	0	ab	6	ab	0	ae	0	ab

Table 30: continued

SITE/REGIONAL	Id-1		Mod-1		Got-2		Dip-1		Gpd-1		Pgm-1		Pgm-2	
Thatcher	1	a	0	a	0	a	0	a	0	a	2	a	2	a
	0	b	0	b	1	b	2	b	1	b	0	e	0	b
	1	ab	1	ab	1	ab	0	ab	1	ab	0	ae	0	ab
Johnson	10	a	14	a	7	a	1	a	1	a	16	a	16	a
	2	b	1	b	7	b	12	b	5	b	0	e	0	b
	3	ab	0	ab	2	ab	3	ab	9	ab	0	ae	0	ab
Lenover	19	a	17	a	6	a	2	a	5	a	23	a	23	a
	0	b	1	b	5	b	20	b	8	b	0	e	0	b
	4	ab	3	ab	12	ab	1	ab	7	ab	0	ae	0	ab
EAST CHATHAM	59	a	67	a	24	a	12	a	13	a	86	a	87	a
	6	b	3	b	25	b	70	b	30	b	0	e	0	b
	20	a	8	ab	38	ab	5	ab	36	ab	1	ae	0	ab
C. Wilkinson	26	a	41	a	26	a	7	a	5	a	45	a	43	a
	2	b	1	b	5	b	36	b	20	b	0	e	0	b
	16	ab	0	ab	14	ab	2	ab	11	ab	0	ae	2	ab
N.R. Wilkinson	3	a	4	a	3	a	0	a	0	a	6	a	6	a
	0	b	0	b	0	b	5	b	2	b	0	e	0	b
	3	ab	2	ab	3	ab	1	ab	1	ab	0	ae	0	ab
James	23	a	19	a	14	a	14	a	7	a	25	a	22	a
	2	b	2	b	0	b	10	b	15	b	0	e	0	b
	0	ab	2	ab	11	ab	1	ab	3	ab	0	ae	3	ab
Leslie	7	a	6	a	8	a	3	a	2	a	10	a	10	a
	0	b	1	b	0	b	5	b	5	b	0	e	0	b
	2	ab	0	ab	2	ab	2	ab	3	ab	0	ae	0	ab

Table 30: continued

SITE/REGION	Id-1	Mod-1	Got-2	Dip-1	Gpd-1	Pgm-1	Pgm-2
WHEATLEY	59 a	70 a	51 a	24 a	14 a	86 a	81 a
	4 b	4 b	5 b	56 b	42 b	0 e	0 b
	21 ab	4 ab	30 ab	6 ab	18 ab	0 ae	5 ab
POOLED SAMPLES	330 a	350 a	231 a	104 a	99 a	455 a	457 a
	20 b	22 b	73 b	339 b	211 b	0 e	0 b
	81 ab	25 ab	163 ab	24 ab	132 ab	12 ae	10 ab

APPENDIX III

EFFECTIVE POPULATION SIZE

Effective population size (N) was calculated from the equation $F=1/2N$, where F is Wahlund's variance (Table 24). Effective population size can also be determined independent of Wahlund's variance. A ratio of populations with fixation (X) at any one locus to the total number of populations (Total) may be determined. The probability of only detecting homozygotes with the fixed allele in the population is the frequency of the fixed allele in the population considered to the power Y. For example, if the frequency of the fixed allele 'a' in a particular population is 0.7000, then the probability of only detecting homozygotes with the 'a' allele is

$$(0.7000)^{Y} = \frac{X}{Total} .$$

Then, Y will be the number of alleles in the population, and Y/2 will be the effective population size.

Using this method, the estimate of mean effective population size based on renal enzyme data is 5 (Table 31) and based on erythrocytic enzyme data is 11 (Table 32). These results corroborate the theory of small breeding unit size. Furthermore, using both methods to calculate N, i.e., dependent and

independent of Wahlund's variance, the estimate of N appeared similar (see Tables 24 and 32).

Table 31: A summary of effective population size, independent of Wahlund's variance, based on renal enzyme data. Only samples consisting of 5 or more animals are included.

	1973				1974				
	Id-1	Mod-1	Got-2	Dip-1	Id-1	Mod-1	Got-2	Dip-1	Gpd-1
X / Total	1/13	2/13	no fixation	1/13	3/29	9/29	1/29	4/29	no fixation
	0.0769	0.1539		0.0769	0.1035	0.3105	0.0345	0.1379	
Frequency of fixed allele	0.6221	0.7518		0.4365	0.8796	0.9131	0.7674	0.7516	
N (approximate)	2.5	3.5		1.5	9	6.5	6.5	3.5	

Mean for N based on 1973 and 1974 renal enzyme data for 4 loci - 5.

Table 32: A summary of effective population size, independent of Wahlund's variance, based on erythrocytic PGM data. Only samples consisting of 5 or more animals are included.

	Pgm-1			Pgm-2		
	1974	1975	1976	1974	1975	1976
$\frac{X}{Total}$	22/35	8/16	15/21	29/35	7/16	12/21
	0.6286	0.5000	0.7143	0.8286	0.4375	0.5714
Frequency of fixed allele	0.9870	0.9596	0.9792	0.9875	0.9756	0.9758
N (approximate)	15	8.5	8.5	7.5	17	11

Mean for N based on 1974, 1975, and 1976 erythrocytic PGM data for 2 loci - 11.

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